Poultry Breeding and Genetics

R.D. Crawford
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Edited by

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FOREWORD

Some readers of this book may well say "with all those pages, why should it need any more just for a Foreword?" And some of the younger readers may even say "if we must endure a Foreword, why should the editor get a doddering old nonagenarian to write it?"

In one way, I can claim that it is partly my book, but I have been spared all the work that the editor, Dr. Crawford has put into its preparation. Here's how that happened.

At the closing session of the Poultry Science Association meetings held at the University of Guelph in 1984, I asked the retiring president (Dr. George Arscott) to see if the PSA would sponsor a new book on poultry genetics.

My *Genetics of the Fowl*, published in 1949, was out of print in the sixties. As I was then mopping up my research in preparation for retirement, I didn't feel that I should undertake a revision of that book. Moreover (I confess) specialists in some branches of poultry genetics had pushed their fields of research so far ahead that I could scarcely understand them. Clearly, as no volunteers had covered the whole outfield, what was really needed was a new monograph with chapters about advances in poultry genetics written by the specialists who had made those advances.

Fortunately the PSA endorsed this idea, and (also fortunately) Dr. Roy Crawford of the University of Saskatchewan was asked to be editor of the new book.

As this is written, I have not seen any chapters of that new book, but Dr. Crawford has kindly provided me with an outline of its five parts and 42 chapters. It is to be 'a reference work; a comprehensive and critical review of literature and current practice in poultry breeding and genetics', and to cover what is known, not only about chickens and turkeys, but also about six other species ranging from Japanese quail to geese and ducks.

One part deals with poultry biology (four chapters), another with qualitative genetics (17 chapters), and a third with immunogenetics, molecular genetics and even the possibility of genetic engineering.

A fourth section is about quantitative genetics and selection, requiring 15 chapters to cover variation in meat traits, in eggs, and in seven related topics ranging from resistance to disease, nutritional requirements, and behavior to heterosis.

The last of the five sections is about applied breeding and selection, and also about breeding special stock for special environments. It covers the needs of industrial breeders, hobbyists, and programs in developing countries.

There is an old saying that "the mills of the gods grind slowly". So does the preparation, editing and publishing of good books on poultry genetics. I hope that I live long enough to see this one in print before I die. When it appears, Dr. Crawford well deserves the thanks of all poultry breeders for his long labor of love with it.

F.B. Hutt

PREFACE

This book has been prepared at the request of F.B. Hutt and the Poultry Science Association as a service to poultry geneticists and to those who work in related areas. Many have felt the need for a current and comprehensive review of poultry genetics literature, which would include new areas of research and discovery, and which would include review of genetics knowledge concerning minor poultry species. The classic treatise on the subject, Hutt's (1949) *Genetics of the Fowl*, has been out of print for many years and it is now inevitably out of date.

Preparation of this book began in 1985. It has been designed as a reference work, and as a comprehensive and critical review of literature and current practice in poultry breeding and genetics. The intended readership is research workers, industrial poultry breeders, graduate students, teachers, and scholars. Contributing authors were carefully selected from the world community of poultry scientists and practitioners, to ensure that particular topics were reviewed by those who are highly qualified to do so.

Eight species of domesticated birds are considered here. They are collectively called 'poultry' in the book title and throughout the text. That term has different meanings in different countries, but here it is used in a broad sense to include all avian species that reproduce freely under the care of man. Similarly, there is variation around the world in terminology for species, sexes, ages, and market categories of poultry. The terminology used here has been made uniform in all chapters to assist the reader; meaning of the various terms will be clear from context. The eight kinds of poultry included here are: chicken or domestic fowl (*Gallus domesticus* or *G. gallus*), turkey (*Meleagris gallopavo*), Japanese quail (*Coturnix japonica*), guinea fowl (*Numida meleagris*), ring-necked pheasant (*Phasianus colchicus*), domestic duck (*Anas platyrhynchos*), muscovy duck (*Cairina moschata*), and goose (*Anser anser* and *A. cygnoides*).

All of the chapters which follow were given peer review by one or more colleagues, with subsequent revision by the authors where needed. Full responsibility for chapter content is that of editor and authors, and ultimately it is the responsibility of the chapter authors. Those serving as peer reviewers were: L.D. Bacon, M.R. Bakst, R.J. Balander, G. Barbato, J. Baumgartner, F.H. Benoff, J. James Bitgood, S.E. Bloom, W.M. Britton, Helene C. Cecil, J.R. Chambers, K.M. Cheng, R.K. Cole, M.M. Compton, R.D. Crawford, V.L. Christensen, L.B. Crittenden, D.M. Denbow, R.W. Fairfull, D.K. Flock, T.W. Fox, G.W. Friars, V.E. Garwood, J.S. Gavora, A.A. Grunder, R.O. Hawes, Edmund Hoffman, W.F. Hollander, R.M. Hulet, G.J. Kraay, M.P. Lacy, S.J. Lamont, A.J. Lee, H.L. Marks, J. Nagai, K.E. Nestor, J.S. Otis, M.P. Richards, T.J. Sexton, A.K. Sheridan, Robert N. Shoffner, J.N.B. Shrestha, J. Robert Smyth, Jr., C.F. Strong, Jr., H.P. Van Krey, K.W. Washburn, Barbara A. West, and S. Wezyk.

All chapter manuscripts were subject to editorial revision to give a modicum of uniformity in presentation, word usage, spelling, grammar, and punctuation across many chapters written by many authors of many nationalities. The language of the book is Canadian — an erratic, irregular, and irreverent blending of proper English and American usage, borrowing freely from French, German, Ukrainian, and many other languages, and from some of the sounds of nature.

Reviewing of published literature requires both library and people resources. A debt of gratitude is owing to libraries and librarians in many places, and particularly to the magnificent James E. Rice Memorial Poultry Library collection at Cornell University

(Samuel G. Demas, director). Many colleagues and associates helped chapter authors in editorial matters; those especially acknowledged by authors were Mary Jane Charles and Cathleen Nichols (Chapter 13); R.L. Kendall and J. Diehnelt (Chapters 19 and 21); S. Cheng, J. Fulton, P. Munns, and E. Steadham (Chapter 22). Secretaries and typists warranting particular thanks from authors were S. Jackson, B. Bliss, Ann Shuey, Diane Colf, and Joy Lusk.

Camera-ready copy for this book was prepared by the Division of Extension and Community Relations, University of Saskatchewan. The process utilized an Apple Macintosh computer and the Microsoft Word 3.01 program, with laser printing in Times typeface. Production was guided and supervised by B. Wolfe, with assistance from J. Greuel. The entire treatise was transcribed from manuscripts to computer by Carol Bahnmann, and she composed the pages and tabular materials — all with astounding speed and accuracy, and with high good humor throughout. The excellence of her work made the editor's task a pleasant one. Graphics were prepared by the Division of Audio-Visual Services, University of Saskatchewan from materials provided by chapter authors. The tedious task of proof-reading was mainly the work of Joan Seed, who also helped considerably in the editorial process. The author index was assembled by Janice Yule. Chapter authors assisted in developing the subject index, which was assembled by the editor and Ralph G. Somes, Jr.

Preparation of camera-ready copy was made possible by an exceedingly generous benefactor grant from Oliver J. Hubbard, Walpole, New Hampshire, U.S.A. His support is very gratefully acknowledged. Additional funding was provided by the Agricultural Research Trust, University of Saskatchewan. Part of the editorial work was conducted during a one-year sabbatical leave from University of Saskatchewan. The editor is grateful to his Department, College, and University for facilitating and encouraging the preparation and completion of the treatise.

R.D. Crawford

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Part I. POULTRY BIOLOGY

Chapter 1. ORIGIN AND HISTORY OF POULTRY SPECIES

R. D. Crawford

The history of poultry species has intrigued scholars for years. They have been interested in identifying the wild ancestors of domestic birds, in the diffusion or spread of a species from one civilization to another and from one territory to another, and in the evolution of poultry species under domestication. The earliest evidence of domestication relies on interpretation of archaeological discoveries of bones and artifacts. Literary records became available with advanced civilizations, but they too require interpretation to modern poultry biology. Recent history is generally available in written form. Zeuner's (1963) review, emphasizing archaeology and classical literature, has been the standard reference work for many years. The more recent review edited by Mason (1984) also considers these aspects, but it stresses biology and evolution of domesticated species. In this chapter and the next, all of these aspects are considered again but in somewhat more detail than found in the reviews of Zeuner (1963) and Mason (1984), both of which gave most attention to mammalian species.

Interest in origin and history of poultry species to the present has always been mostly academic. Unlike plant breeders, poultry breeders have made little effort to seek genetic improvement through use of wild ancestors or primitive relatives of modern stocks. However, with rapid advances in genetic engineering, it soon will be feasible to use such resources. And then a knowledge of origins and history may have practical value.

Sequence Of Livestock And Poultry Domestications

The sequence of mammalian domestications is well-known (Zeuner, 1963; Mason, 1984). The ranking of poultry species domestications within this sequence is of interest here. Details and references for each species will be given later in this chapter. Dogs were the first, near the end of the Pleistocene and the beginning of the Recent geological periods. Precise dates of domestication are not known, but they lie somewhere in the interval of 12,000-10,000 B.C., seemingly occurring in many separate places in the Northern Hemisphere, while man was a hunter-gatherer. The next to be domesticated were sheep and goats, in the 'fertile crescent' of southwest Asia beginning about 9000 B.C. Man was still nomadic but he could manage grazing animals. Domestication of plants began about 7000 B.C. and it was then that man began establishing permanent settlements. Pigs were domesticated about that time (7000 B.C.), also in southwest Asia. Cattle did not appear as part of man's possessions until about 6000 B.C., first in southwestern Asia and southeastern Europe, and spreading slowly elsewhere to evolve into the humpless shorthorn by 3000 B.C., the cervico-thoracic humped zeboid by 4000 B.C., and the thoracic humped zebu by about 2500 B.C.

Very recent archaeological discoveries in China indicate that chickens had been domesticated by 5400 B.C.; people of the Cishan Culture had chickens then, along with dogs and pigs. But it is not known whether these birds made much contribution to modern domestic fowl. Chickens from the Harappan Culture of the Indus Valley (2500-2100 B.C.) may have been the main source for diffusion through the world.

Domestication of horses and donkeys was surprisingly late. The first evidence for horses dates from about 3500 B.C. in the Ukraine, and for donkeys from about the same time in northeast Africa.

Geese and mallard ducks were probably next in the sequence. They are known from Chinese archaeological discoveries dated at about 2500 B.C. A separate domestication of geese was fully established in Egypt by 1500 B.C., and perhaps also in Europe. There appears to have been no successful local domestication of mallard ducks in the west until the Middle Ages.

Ring-necked pheasants were taken into captivity by the Greeks about 1300 B.C. Domestic guinea fowl from East Africa were also known in the Greek civilization, but modern stocks have probably descended from West African birds introduced into Europe by Portugese explorers about A.D. 1500.

Domestication of turkeys and muscovy ducks in the Americas is believed to have been very recent. The earliest evidence for turkey domestication in Mexico is from the period 200 B.C.-A.D. 700 when cultivation of plants was already highly developed. The Spanish Conquistadores found muscovy ducks fully domesticated in Columbia and Peru during the 16th century. They differed very little from the wild birds, perhaps indicating very recent acquisition.

The latest domestication of a poultry species is probably that of Japanese quail, which occurred about the 11th century in Japan, China, or Korea.

Effects Of Domestication

The changes in a species which occurred during and after domestication have been examined by many, particularly in mammals (Zeuner, 1963 and Mason, 1984), but also in birds (Sossinka, 1982). All have suggested that preadaptation or predisposition to domestication had importance.

Sossinka (1982) listed four traits that are common to most domesticated bird species. First, in early stages of domestication birds would have to forage on their own, favoring species that were seed or grass eaters. Second, they had to be able to reproduce in captivity, giving an advantage to species that were not highly dependent on specific climatic and environmental factors for breeding. Third, the ability to imprint was important in initial taming. And finally, the species had to have a social order that permitted the keeping together of large numbers of individuals.

A usual immediate consequence of mammalian domestication was a decrease in body size, probably due both to inbreeding and to inadequate husbandry. Poultry species seem not to have been affected in this way. Hence in archaeological studies, bones larger than the wild-type for a species have been taken as proof of domestication. The only exception seems to be in turkeys, where Mexican domestic birds of the 16th and 17th centuries were much smaller than wild turkeys.

A second consequence of domestication in mammals was accumulation of coat color variations, particularly involving leucism. This has been noticeably characteristic of domesticated birds, and especially so in chickens.

In domesticated mammals, a usual finding has been alterations in facial structures and dentition. Similar modifications seem not to have occurred in birds. However alterations to the limbs involving changes in length, in muscle attachments, and in joint structure are evident in both domesticated mammals and birds.

Changes in skin covering, muscling, fat deposition, and brain size were later accompaniments to domestication in both mammals and birds. Such changes and modifications are particularly evident in species where great diversity has been accumulated, as for instance in chickens, but they can also be demonstrated in species such as guinea fowl and muscovy ducks which still closely resemble the wild ancestor. Many less noticeable changes, for instance changes in physiology and behavior, accumulate over time in all species. The description and interpretation of these by Sossinka (1982) should be consulted.

Purpose And Utilization Of Domesticated Birds

Zeuner (1963) has discussed in detail the various origins and stages of domestication of mammals and the uses made of them by man. Most species were used from the beginning as a food source, although some (dogs, cattle, horses) primarily served a role as work animals.

The situation with domesticated birds was quite different. The archaeological and historical records indicate that for nearly all bird species, the first use of domesticated stocks was cultural - in religion and superstition, in decorative arts, and in entertainment; only much later were they utilized as a source of human food. Chickens especially followed this sequence, but it is clearly evident in the history of others. For instance, geese in ancient Rome were held sacred but they had also become a food delicacy; turkeys were in transition from cultural use to food use when the Spanish discovered them in the New World; Japanese quail were kept for centuries as caged song-birds and only in recent decades have they been exploited as a source of eggs and meat.

CHICKEN OR DOMESTIC FOWL

Most scholars believe that chickens were domesticated from the red junglefowl (*Gallus gallus*) in the Indus valley about 2000 B.C. From there they gradually spread to the east, and to the west from where they eventually encircled the globe.

However, not all scholars accept the majority view. Some believe that the other three wild *Gallus* species may have contributed to the domestic bird. Others believe that southeast Asian chicken stocks were domesticated separately, and may have been derived from one or more extinct species of junglefowl. And others hold the view that there was one very early domestication in Burma, diffusing in all directions, with first archaeological evidence appearing in China and the Indus valley.

Archaeological discoveries in China and elsewhere, and new appraisal of fossil *Gallus* specimens from Europe, encourage a reassessment of chicken origins and dispersal.

Fossil *Gallus* Species

The fossil record for *Gallus* is scanty. Only three specimens, all of them from Europe, have been assigned to the genus (Harrison, 1978). Two specimens are from England. One is a coracoid bone from the Cromerian Interglacial period of the early Middle Pleistocene. The bone has greatest resemblance to red junglefowl (*Gallus gallus*) but it is sufficiently different that it has been assigned to a new fossil species *G. europaeus*. The second fossil is a radius, from the Ipswichian Interglacial period of the Upper Pleistocene. It closely resembles *G. gallus* but because of its very great age it has been assigned to the fossil species. A third fossil, almost certainly belonging to *Gallus*, was described from the Lower Pliocene of Greece and the Black Sea region. It consists of a tarsometatarsus much like *G. gallus* in size and a coracoid that is much longer. It has been assigned to a separate species, *G. aesculapi* Gaudry 1862.

Harrison (1978) has tried to interpret these fossil discoveries, in relation to *Gallus* evolution and distribution, using his theory on the effects of glaciation in the late Pliocene and early Pleistocene on bird speciation. A detailed discussion of the general hypothesis, with examples from many bird species, is given in Harrison (1982). During periods of glaciation bird species which had comprised a single population across the Eurasian continent became fragmented into isolated populations in warm southern refuges. In many species, an eastern and a western refuge was used. For some forest species there were three subpopulations formed - a western one in the Mediterranean or Middle East, a central one in India, and one in far eastern Asia. When glaciers retreated, western and eastern subpopulations would expand northward and eventually they would meet; depending on length of isolation, they might meanwhile have evolved into separate species or subspecies. But the central subpopulation in India would be prevented from expanding northward by the Himalayas and the Tibetan plateau and it would remain in the Indian subcontinent.

Harrison (1978) has applied this speciation theory to the question of *Gallus* evolution and distribution. According to him, presence of fossil *Gallus* in Europe indicates existence of a western refuge subpopulation. It may have gone to extinction during a later glaciation, or it may have been exterminated by man. There is no evidence that it existed into historic times. He cautioned that ancient *Gallus* discoveries in Europe, which have been interpreted to be domestic fowl, should be reassessed; they may actually represent fossil wild *Gallus*.

Presumably an Indian refuge subpopulation of junglefowl has persisted as modern *G. gallus* and related species (Harrison, 1978). It has remained isolated there because of geographic barriers to the north.

The possibility of a third refuge subpopulation in far eastern Asia leads to speculation. Harrison (1978) suggested that survival in a refuge might depend on absence of other species competing for the same ecological niche, and the large complex of pheasant species in Asia may have prevented *Gallus* from surviving there. However, there has been recent discovery of archaeological evidence for chicken domestication in China as early as 5400 B.C., thousands of years before apparent domestication in India (Chow, 1984). Furthermore, some scholars believe that modern Asiatic chickens are so different from *G. gallus* domesticates that they must have had a separate origin (Plant, 1986). It may eventually be shown that a separate Asian species of *Gallus* evolved in a far eastern refuge during glacial periods, and that it persists now in the Chinese chicken breeds. Indeed, Harrison speculated in this way in a personal communication cited by Plant (1985) and in correspondence with the author (Harrison, 1988, personal communication). He, like others, urges that molecular genetics studies be conducted to help resolve the issues.

Harrison's (1978, 1982) speciation hypothesis has been amply proven for temperate bird species. But it should be cautioned that junglefowl are tropical, and his theory has not been proven for tropical species (West, 1988, personal communication).

Wild *Gallus* Species

Four species of junglefowl are known to modern ornithology. One of these, the red junglefowl (*G. gallus*), has without doubt been a major contributor to domestic fowl. It is less certain whether the other three species are ancestral to chickens.

Classification. Junglefowl have been classified by Delacour (1977), Howard and Moore (1984), and Johnsgard (1986) as order Galliformes, suborder Galli, family Phasianidae, subfamily Phasianinae, tribe Phasianini, genus *Gallus*. The four recognized species are red junglefowl *G. gallus* (Linnaeus) 1758, grey or Sonnerat's junglefowl *G. sonnerati* Temminck 1831, Ceylon junglefowl *G. lafayettei* Lesson 1831, and green junglefowl *G. varius* (Shaw and Nodder) 1798. There are no subspecies of grey, Ceylon, or green junglefowl. Geographic variation is very marked in red junglefowl, particularly among males, and this has been recognized by designating five subspecies: Cochin-Chinese red junglefowl *G. g. gallus* (Linnaeus), Burmese red *G. g. spadiceus* (Bonnaterre), Tonkinese red *G. g. jabouillei* Delacour and Kinnear, Indian red *G. g. murghi* Robinson and Kloss, and Javan red *G. g. bankiva* Temminck.

Species nomenclature has undergone some confusing changes (Hutt, 1949). Red junglefowl were formerly called *G. bankiva* and *G. ferrugineus*. The Ceylon junglefowl was once named *G. stanleyi*. And the green junglefowl is probably the one called *G. furcatus* in the early literature.

Description. There are three major monographs on Phasianidae which include detailed description of junglefowl. The old classic is Beebe's treatise (1918-1922), which is now readily available only in its condensed form (Beebe, 1936). Delacour's (1977) treatment emphasizes avicultural information. The monograph by Johnsgard (1986) seeks to emphasize naturalistic ecology and breeding biology.

The special feature which distinguishes *Gallus* from all other Phasianidae is the comb, and the wattles associated with it. In red, grey, and Ceylon junglefowl the comb is a single upright serrated blade and there is a pair of wattles. Head furnishings are not as large and prominent as they are in domestic fowl; in wild females they seldom project beyond the feather coat. The green junglefowl is quite different; the comb is unserrated and multicolored; there is only one median wattle.

Males of all four species have spectacular coloring enhanced by modifications in feather morphology; females of all four have nearly identical feather morphology and they differ only a little in coloring, all of which is drab (Morejohn, 1968a).

The red junglefowl has been proposed as the wild-type standard for mutations found in domestic fowl (Jaap and Hollander, 1954) and that has been generally accepted by geneticists. Feather morphology of wild and domestic forms is essentially the same. Feather color of red junglefowl has been retained almost exactly in the black-breasted red phenotype of domestic fowl, as for instance in Brown Leghorns. A feature which distinguishes red junglefowl males from those of other species is the patch of pale downy aftershafts at the base of the tail coverts; the patch is very evident during some behavioral displays (Morejohn, 1968a). The five recognized subspecies differ in color of ear lobes which range from white to red, in shape and length of neck hackle feathers of males, and in shade of red in male plumage which varies from golden yellow to mahogany.

Ceylon junglefowl may represent an offshoot of the red species which developed in total isolation in Sri Lanka. Male plumage is much like that in red junglefowl except that breast feathers are pointed and fringed rather than being rounded and female-like, and they are red rather than black. Males of this species have a peculiar patch of bluish-purple feathers on the upper breast. Plumage of female Ceylon junglefowl is similar to that of the red species.

Plumage of the grey junglefowl male is quite different from that of the other species. The main distinguishing feature is 'sealing wax' spots on some of the feathers. The spots are actually expanded and flattened portions of the rachis. They are arranged in a series along the rachis, those that are subterminal being white, and the terminal spots being shredded and yellow. They occur principally in the neck hackle, saddle, and wing coverts. Most of the body feathers are black with a white shaft and a grey border. Wing and tail feathers are black. Grey appearance of the bird, rather than red as in *G. gallus*, led Hutt (1949) to suggest that the two species differed with respect to silver and gold alleles, but this was refuted by Kimball (1954) and Morejohn (1968a) who found that in *gallus* x *sonnerati* hybrids grey coloration did not exhibit simple dominance over red as might have been anticipated. Plumage of the grey junglefowl female differs from that of the red species principally in breast feathers which are white with broad black or brown borders.

The green junglefowl is regarded as the most primitive of the four species. Distinguishing features of the plumage are the presence of 16 tail feathers rather than 14 as found in the other species, and short truncate neck hackle feathers in males rather than the long pointed hackle feathers of the other species. Plumage of the male is mostly glossy black, but hackle and saddle feathers particularly are edged with bronze and yellow imparting a distinctly green coloration to the entire bird. In females, feathers of the back and rump are pencilled much like that in Dark Cornish chickens, upper breast feathers have a dark edging, lower breast feathers are pale, and the rest of the plumage exhibits irregular barring.

Males in three of the junglefowl species (red, grey, Ceylon) exhibit an eclipse plumage after the breeding season; green junglefowl do not (Morejohn, 1968a). Molting involves only the cervical feather tract of the neck in which long pointed hackle feathers of the usual male plumage are replaced by short rounded feathers like those of females. The eclipse plumage persists for only a few weeks, followed by the complete annual molt involving all feather tracts. An eclipse molt occurs in females too but it is not noticed because feather morphology remains the same.

All species are sedentary and omnivorous. Behavior is essentially like that in domestic fowl, but vocalizations differ considerably among species. Red, grey, and Ceylon junglefowl prefer forest habitat and forest clearings. Green junglefowl prefer seashore and rocky scrubland bordering cultivated land. Relative body size data and reproductive parameters are summarized in Table 1.1.

Junglefowl are kept in many zoos and they are popular among aviculturists. But the total numbers in captivity are surprisingly small. Data from the 1982 World Pheasant Association census (Johnsgard, 1986) indicate the following: red junglefowl 1072, grey 432, Ceylon 213, green 82. All species appear to be surviving well in the wild since they readily adapt to man-made alterations in habitat.

Distribution. The red junglefowl has the largest natural range of the four species. There is marked geographic and clinal variation, especially in male plumage, recognized by division into five subspecies. It occurs naturally from Pakistan eastward to China, Hainan, and in eastern India, Burma, and most of Indo-China, and on the islands of Sumatra, Java, and Bali. The grey junglefowl occurs in western and southern India; it is sympatric with red junglefowl at boundaries of its range. The Ceylon junglefowl is restricted to the island of Sri Lanka. The green species is found in Java and on the chain of islands eastward; it is sympatric with red junglefowl in Java, but the two occupy different habitats. Details of

Table 1.1. Body size and reproductive traits of *Gallus* species.

	gallus	*sonnerati*	*lafayettei*	*varius*
Adult weight [1,3],				
males, g	800-1360	790-1136	790-1140	454-795
females, g	485-740	705-790	510-625	m & f
Sexual maturity[2], captive, yrs.	1-2	2	2	1-2
Clutch size[1]	4-8	4-8	2-4	6-10
Egg size[1], mm	45.3x34.4	46.0x36.5	46.3x34.5	44.5x34.5
Egg shell color[1]	white to rosy cream	white to rosy buff	pinkish to buff, brown stippled	buffy white
Incubation time[1], days	19-21	20-21	20-21	21

[1] Delacour (1977)
[2] Johnsgard (1986)
[3] Long (1981)

natural range are given by Delacour (1977), Johnsgard (1986), and Long (1981).

Red junglefowl have been successfully introduced in many places (Long, 1981) sometimes as a wild bird and sometimes as a semidomestic which became feral. Junglefowl of the Philippines, Micronesia, Melanesia, and Polynesia are all considered to be introduced feral *G. gallus*. Many introductions elsewhere — Australia, New Zealand, West Indies, United States, Hawaii — persisted for a while but never became firmly established. Foreign introductions of other junglefowl species appear not to have been successful.

Monophyletic Or Polyphyletic Origin

Were domestic fowl derived exclusively from red junglefowl (*G. gallus*) or did other species, extinct or living, also make a contribution? Research and debate on this question have filled many pages of the literature. The question is still not fully resolved.

Darwin (1896) gave detailed consideration to the issue and concluded that the red junglefowl was the only ancestor. Most naturalists agreed with him. But there was no agreement among writers of poultry treatises of that era. They debated fiercely and sometimes vindictively through multiple editions and reprintings of their books - Dixon, Moubray (a pseudonym), Main, Tegetmeier, Wingfield and Johnson, Wright (see Chapter 2). In his excellent historical account of cock-fighting, Finsterbusch (1929) was adamant that game cocks arose from Malay fowl, the *G. giganteus* of Temminck, and not from bankivoid fowl (*G. gallus*), the ancestor of 'dung-hill' or ordinary chickens. Recently Plant (1986) has resurrected arguments from the old masters in championing the view that modern Asiatic breeds (Brahma, Cochin, Langshan) have a different origin from bankivoids and Malays. Harrison's (1978) interpretation of fossil *Gallus* should not be overlooked. Hutt

(1949) reviewed the available evidence and found it all to be inconclusive. He recommended, perhaps in jest, that the zoological name for the domestic fowl should "depend somewhat upon the particular faith of the writer who uses it"; those believing in monophyletic origin should use *Gallus gallus*, and those favoring polyphyletic origin should label them *Gallus domesticus.*

To resolve the problem, Punnett's (1923) *Heredity in Poultry* recommended systematic matings among the four junglefowl species and with domestic fowl breeds; "until this has been done there can be no certainty as to the manner in which our various races of poultry have arisen, and any further discussion as to their origin is merely waste of time". In Hutt's (1949) *Genetics of the Fowl* it says: "this is as pertinent now as when it was first written". Not much has changed in the ensuing decades.

However, a brief consideration of the literature on hybrids may be instructive. There is a large body of literature, most of it summarized by Gray (1958). But it is not very helpful in genetic considerations. Most of it is based on avicultural observations, seldom extending beyond production of F_1 progeny. Hybrids have been obtained from crosses among all junglefowl species and with chickens. But when performance of these hybrids has been measured in backcross and *inter se* matings, the results have usually been disastrous. Morejohn's (1968b) study of hybridizing between captive red (*G. gallus*) and grey (*G. sonnerati*) junglefowl is illustrative. His data are summarized in Table 1.2. By utilizing special rearing conditions and longterm habituation, he was able to obtain a cross of grey male with red females, but he could not obtain the reciprocal. F_1 females were backcrossed to males of both species, and F_1 birds were mated *inter se.* Experimental design was less than ideal and difficulty was encountered in providing adequate hatching conditions for grey junglefowl embryos, but overall his results revealed major barriers to introgression beyond the production of F_1 hybrids. A large number of F_1 hybrids was produced; they were robust and easily raised. In the F_2 generation there was very high mortality at all stages of incubation, and many embryos were grossly defective; only five F_2 chicks hatched and all of them died soon after. The backcross chicks, although very few in numbers, were vigorous; none of the many backcross embryos which died during incubation exhibited gross anatomical defects. But it remains a matter of conjecture whether any genomic trace of hybrid origin could persist for long in a backcrossed population, in view of the total lethality of the F_2 generation. Only in the case of hybridizing between red junglefowl and domestic fowl does the literature reveal complete compatibility (Gray, 1958). All of the other hybrid combinations warrant further study.

The disciplines of biochemical genetics, immunogenetics, and molecular genetics are providing help in resolving the question of monophyletic or polyphyletic origin. Pertinent research includes Baker's series of papers on molecular genetics of egg white proteins (Baker, 1964, 1968; Baker and Manwell, 1972; Baker et al., 1971). All of her work supports the view that red junglefowl (*G. gallus*) were the sole or main ancestor of domestic fowl. The possibility of introgression from grey junglefowl (*G. sonnerati*) was especially investigated but no positive evidence of such was found in egg white protein data from a number of breeds; neither were the distinctive *G. sonnerati* proteins found among indigenous chickens of southern India where grey junglefowl are native.

Weiss and Biggs (1972) have demonstrated that the red junglefowl (*G. gallus*) from Malaya carries a genetically transmitted endogenous retrovirus genome similar to that in domestic fowl. Subsequently Frisby et al. (1979) showed that other species of junglefowl lack detectable retrovirus sequences homologous to those of chickens. Their interpretation was that the RAV-O genome had been introduced into the germ line of *G. gallus*

Table 1.2. Fertility and hatchability in matings of *Gallus gallus* and *G. sonnerati*. (Adapted from Morejohn, 1968b).

Mating Male Female	Eggs set, no.	Eggs fertile, no.	Hatched, no.	Embryos dead, %
Grey x grey	45	27	8	70.4
Red x red	333	196	98	50.0
Grey x red	115	78	61	21.8
Grey x F_1	28	23	2	91.3
Red x F_1	28	23	1	95.7
F_1 x F_1	321	227	5	97.8

following speciation but before domestication. It would be of interest to extend these studies to a much broader array of domestic fowl breeds and strains and genomes. Further clues to origins and descent might be revealed thereby.

Origin And Early History

There is still no clear answer to the questions of where and when chickens were first domesticated. An opinion overlooked in most reviews is that of Peters (1913) who believed that the original site of domestication was in the region of Burma. His opinion was based on comparative ornithology and earlier views of others, but he had neither literary nor archaeological evidence to support this conclusion. Darwin (1896) expressed a similar belief. And now West and Zhou (1988) have provided strong supportive argument.

Two places and times have yielded solid evidence for the presence and keeping of chickens. Were these the places and times of domestication, or were those chickens derived from an earlier domestication somewhere else?

Chickens in the Harappan culture of the Indus Valley. Archaeological evidence (Zeuner, 1963) indicates that chickens were being kept by people of the Harappan culture (2500-2100 B.C.) of the Indus valley. Many have taken this as evidence that chickens were first domesticated there, although recent discoveries in China and elsewhere may prove this to be incorrect. It is likely however that the Indus valley was a very major staging point for the diffusion of chickens both east and west and eventually around the world.

The Harappan culture was the first urban civilization on the Indian subcontinent. It occupied a huge area of the Indus valley and plain in present-day Pakistan. The twin capital cities of Harappa and Mohenjo-Daro are the best known archaeological sites. Agriculture was the economic base for the Harappan culture, and there was an extensive trade network with Sumeria and Mesopotamia.

Zeuner (1963) reviewed the archaeological evidence. Seals were found at Mohenjo-Daro depicting fighting cocks. Various clay figurines of chickens were also found including one of a hen with a feed dish. Chicken bones were much larger than those of junglefowl, indicating selection for increased size. Two clay figurines were found at Harappa, appearing to represent a cock and a hen. Based on all of these, Zeuner (1963) estimated that full domestication of chickens had taken place by about 2000 B.C. Without referring directly to the Harappan culture, Wood-Gush (1959, 1985) estimated a much earlier date of 3200 B.C.

Domestic fowl in ancient China. Exciting archaeological discoveries have been made very recently in China which indicate that domestic fowl were kept there as early as 5400 B.C., several thousand years before the apparent domestication of chickens in the Indus valley. Short descriptions of some discoveries have been given by Ho (1977) and Watson (1969). Plant (1985) has reviewed and reprinted an English translation of parts of a 1981 paper by Zhou Ben-Xiong (also known as Chow Ben-Shun) concerning discoveries at the Cishan site. Chow (1984) has published an English-language review of the discoveries, and they have also been reviewed by West and Zhou (1988).

The archaeological site of Cishan, in Wu'an county of Hebei province, has revealed a primitive farming culture dating from 5900-5400 B.C. Chow's (1984) interpretation of animal remains found there is that dogs, pigs, and chickens were domesticated, and that chicken domestication may have occurred as early as 6000 B.C. Most of the chicken remains were tarsometatarsal bones (Zhou, 1981, in Plant, 1985). Thirteen were considered to be from males, because of their size and presence of a spur, and a smaller bone without a spur was from a female. Their measurements were greater than those for red junglefowl and less than those for modern domestic fowl. Clavicle, humerus, femur, and radius and ulna specimens were also found. Both high frequency of chicken bones in the animal remains and greater size than in junglefowl were considered as positive evidence for domestication. Similar bone discoveries from the same time period were made at the Pei-li-Kang site in Henan province (Chow, 1984).

The Yang Shao culture of south Shensi, dating from 4800-3000 B.C., has also yielded evidence of domestic fowl. Watson (1969) reported that chicken bones were found at Pan-p'o in Sian, but he cautioned that their interpretation as domesticated birds needed confirmation, although Chow (1984) accepted them as domestic. Zhou (1981, in Plant, 1985) drew attention to similar chicken bone remains at another site dating to the fifth millenium B.C., and to the finding of earthenware models of chickens and of chicken bones at sites dating to the third millenium B.C. According to the review by West and Zhou (1988), 16 Neolithic sites in northern China have now yielded evidence of domestic fowl, all of them predating Mohenjo-Daro by many centuries.

Ho (1977) made very brief mention of chicken remains at several sites, interpreting these as evidence that junglefowl had much wider distribution in ancient China than they do at present, and presuming that domestication had occurred locally. Crawford (1984) supported this interpretation. However West and Zhou (1988), using information from Ho (1977) and from other sources, have shown that the environment at these Neolithic sites has been one of semiarid steppe for at least the past 10,000 years and hence not likely to have been natural territory for junglefowl. They proposed instead that domestication had occurred somewhere in southeast Asia and chickens were taken by man from there to northern China by about 6000 B.C.

Up to now, the accepted view has been that of Darwin (1896), whose date of 1400 B.C. for introduction of chickens from India to China has been widely quoted. That date was derived from translation of portions of a Chinese encyclopedia published in 1609 and derived in turn from ancient documents. Brown (1929) gave the date of introduction as 1500 B.C. when Buddhist priests carried chickens from India to China. The discovery of chicken bones at Neolithic sites in northern China now destroys that accepted view.

Archaeological discoveries elsewhere. West and Zhou (1988) have assembled an astounding list of chicken osteological evidence from securely dated archaeological sites in Europe and Asia. In addition to the 16 discoveries in China predating Mohenjo-Daro in the Indus valley (2500-2100 B.C.), 13 sites have now been identified in Europe and western Asia also yielding chicken bones older than those of Mohenjo-Daro. These include sites in Iran, Turkey, Syria, Greece, Romania, and the Ukraine. Eight other sites in China, Turkey, Syria, Spain, Romania, and the Ukraine have yielded osteological evidence that is contemporary with that of Mohenjo-Daro. Considering all of this evidence, West and Zhou (1988) conclude: "if the European and Western Asian evidence offers a serious challenge to the Indus Valley civilization as the ultimate source of domesticated fowl, the evidence from China topples it altogether".

Scholars should now examine the osteological evidence assembled by West and Zhou (1988) to verify it, and to place it in context with the abundant literary evidence which until now has been interpreted on the assumption that Mohenjo-Daro was the starting point for diffusion of chickens throughout the world. That assumption may no longer be tenable.

Diffusion westward from the Indus Valley. All recent reviews of chicken history have begun with Mohenjo-Daro (Crawford, 1984; Wood-Gush, 1959; Zeuner, 1963). The earlier classical treatises of Hehn (1885) and Peters (1913) were written long before discovery of the Indus valley civilization, but they too gave most attention to diffusion into southwestern Asia and Europe from the east. Aldrovandi's chapter on chickens (translated by Lind, 1963) presents a remarkable review of classical literature and knowledge up to 1600 A.D. in Europe, all of it indicating eastern origin.

Presuming that the major western distribution centre for chickens was the Harappan culture of the Indus valley, it might be expected that diffusion outwards would occur gradually. Where the birds were absorbed by advanced civilizations there should be both archaeological and written record, and from pagan areas between there would be no record at all. Carter (1971) considered diffusion by both land and sea routes, and using dates of first records in widely separated places he estimated a diffusion rate of 1.5-3 km per year, which is reasonably consistent with rates for other things, techniques, and ideas.

Within the Indus valley, indications are that chickens were first used for sport and not for food (Zeuner, 1963). The Aryans invaded India about 1500 B.C. and soon included chickens in their culture, but they did not enjoy them as a food. By 1000 B.C. chickens had assumed religious significance and eating of them was forbidden.

Persia and Mesopotamia. Introduction of chickens to Persia (Iran) may not have occurred until the first millenium B.C. since Brown (1906, 1929) stated that the Persians brought fighting cocks home after their conquest of India about 537 B.C. However, chickens must have been well-known there much earlier since in the Zoroastrian literature of the Kianian Period (2000-700 B.C.) the cock was known as the Herald of the Dawn (Wood-Gush, 1959) and it also played a role as guardian of good against evil (Zeuner, 1963). West and Zhou (1988) list a bone discovery from Iran dated at 3900-3800 B.C., and two others that correspond with the literary record.

The Persians carried chickens westward to Mesopotamia and Asia Minor (Zeuner, 1963). The Medes played a prominent role, leading later to Greek reference to chickens as the Median bird. Seals from the eighth century B.C. depict chickens in a religious context, and cock-fighting remained very popular. First introduction could have been very much earlier, since West and Zhou (1988) list several bone discoveries in Turkey and Syria that predate Mohenjo-Daro.

Greece. Although there is osteological evidence for chickens in Greece predating the Harappan civilization (West and Zhou, 1988), the rich literary record does not support the continuing presence of the species from prehistoric times. Pollard's (1977) account of birds in Greek life and myth indicates that chickens reached Greece from Persia in the seventh century B.C. They were not mentioned by Homer and Hesiod from about 900 B.C. and presumably were unknown then. The first literary reference is in Theogenis who wrote in the latter half of the sixth century B.C., and many others followed including Aristophanes and Aristotle. According to Zeuner (1963) acceptance of chickens was very rapid and they soon became widespread, even in outlying Greek colonies in Sicily and southern Italy. Chickens had great importance in sport, but they also assumed major importance in religious and cultural symbolism. Except among the poor, they were not much exploited as a food even though there was no taboo against eating them. Distinctive breeds or types were already in evidence (Pollard, 1977). Both Plato and Aristotle distinguished between high-bred and low-bred chickens, egg production of high-bred birds being inferior. Two breeds were recognized from Tanagra near modern Athens; one was for fighting, and the other was black with crest and wattles 'like anemones', which could refer to the rose comb mutant. By the second century A.D. Ptolemy knew of a bearded chicken which probably was the muffs and beard mutant.

Egypt. Chickens became firmly established in Egypt under Greek and Persian influence (Coltherd, 1966). They had been present in Egypt and in Crete many centuries earlier, presumably representing trade in exotic creatures, but they had not persisted. Those very early occurrences have created both controversy and puzzlement in assembling an historic record of chicken diffusion, and so they will be described in detail.

Lowe (1934) drew attention to a steatite seal from Crete, illustrated in Sir Arthur Evan's *Cretan Pictographs and the Pre-Phoenician Script* published in 1895. The seal is dated from about 1500 B.C. and clearly depicts a rooster.

Several pictorial and written artifacts, all described by Coltherd (1966), have been found in Egypt. The 'chick' hieroglyph found in the earliest inscriptions is now thought to represent a guinea fowl or a quail and not a chicken. But among graffiti on blocks from a temple of the Middle Kingdom near Thebes, dated at about 1840 B.C., there is a fairly definite representation of a chicken. The royal annals of Thutmose III (1479-1447 B.C.) include a list of tribute from an Asian land; the phrase 'four birds of this country; they bear every day' most likely pertains to chickens. Perhaps of more importance are three pictorial records, all securely dated, and all unmistakably representing chickens.

One of these is a rooster's head in a mural from the tomb of Rekhmara, vizier of Thutmose III, at Thebes. It is dated from about 1450 B.C. Lowe (1934) has provided a detailed description. The mural was discovered about 1835, and drawings of it appeared in several books on Egyptian art and archaeology. The first reproduction was in Hoskins' *Travels in Ethiopia* published in 1835. Subsequent reproductions of the mural indicated its rapid deterioration since the rooster's head was no longer clearly defined. The mural depicted a procession of fifty human figures representing many races bringing tribute to Thutmose III. The tribute included a variety of animals - monkeys, lions, leopards, giraffes, and antelope. Among them was a gold image of the head of a rooster (Figure 1.1). Head furnishings and facial features are very startling. The comb appears to be that of a pea comb heterozygote, and the facial features are those of modern Asiatic breeds (Crawford, 1984). If this interpretation is correct, then the pea comb mutation must be a very ancient one, and Asiatic breed characteristics must have developed soon after domestication of junglefowl.

A second pictorial record of chickens in ancient Egypt is that of a rooster painted on an ostracon, found in association with the tomb of Tutankhamun and probably contemporary with his burial in 1338 B.C. The ostracon and circumstances of its discovery have been described by Lowe (1929, 1934). Except for the very large comb, the rooster very closely resembles wild *Gallus gallus*. It is redrawn in Figure 1.1.

The third piece of pictorial evidence from ancient Egypt is a silver bowl from the reign of Seti II (1200-1194 B.C.) engraved with the figure of a rooster (Coltherd, 1966).

All of these can be interpreted now as a record of exotic trade, and not as evidence that chickens were being kept then in ancient Crete and Egypt (Crawford, 1984). Maritime trade was flourishing at that time, especially involving the advanced civilization of the Egyptians who had extensive trading connections with many parts of Europe, Africa, and Asia. No doubt the chickens and junglefowl of southeast Asia and the Indian subcontinent were part of that trade. Soon thereafter Egypt suffered a political decline and recession in trade. No trace of chickens has been found from the succeeding five centuries. It was not until about 600 B.C. that chickens once again appeared in the written and pictorial records, where they have remained to the present.

Palestine. Chickens were known in Palestine beginning in the seventh century B.C. (Taran, 1975), at about the same time as they reappeared in Egypt. All indications are that the birds were used exclusively for fighting. The osteological record for Israel and Jordan (West and Zhou, 1988) is in agreement with Taran (1975).

Wood-Gush (1959) has stated that chickens did not reach Palestine until about 200 B.C. which is consistent with lack of any mention of them in the Old Testament. Finsterbusch (1929) drew attention to I Kings 4:23, an account of provisions for Solomon's table which included 'fatted fowl'. He interpreted these to be chickens, but Shulov (1985) gave their zoological identification as swans (*Cygnus* spp.). The most scholarly discussion of this passage and two others from the Old Testament is that of Peters (1913), who concluded that there is indeed no mention of chickens anywhere in the Old Testament although references in the New Testament are frequent.

Roman Empire. It has been assumed that the Romans acquired chickens from the Greek colonies in the south of Italy, but Hyams (1972) and Zeuner (1963) both noted the

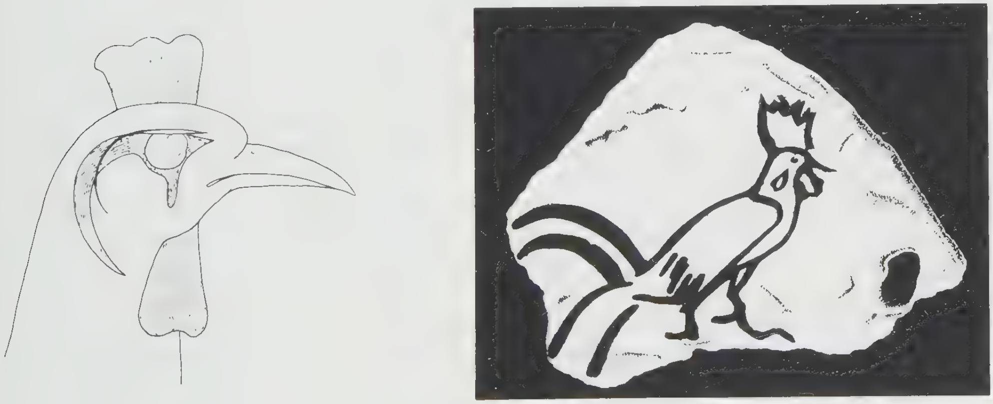

Figure 1.1. Rooster's head in a mural from Rekhmara's tomb, 1450 B.C. (left). Rooster painted on an ostracon from Tutankhamun's tomb, 1338 B.C. (right). (Redrawn from Lowe, 1929, 1934).

possibility of a separate introduction from the north. By the first century B.C. poultry keeping had become a scientific craft according to treatises on the subject by Columella and Varro. These and other writings were extensively reviewed by Aldrovandi (Lind, 1963). The Romans knew about force feeding, hybrid vigor, caponizing, and even about sperm competition. They had access to many breeds or types. They adopted chickens as a food source almost immediately, but they also enjoyed cock-fighting, and they used chickens very extensively in religion, superstition, and divination.

Excavation of the Roman town of Gorsium in Pannonia (Hungary), dating from A.D. 50-350, has provided an intriguing glimpse at animal husbandry during Roman times (Bökönyi, 1984). Gorsium was a military post at first and later it became an administrative and economic centre. It eventually ceased to exist as a town and in modern times it was pasture and crop land, so that nearly complete excavation was possible. A vast array of nearly 50,000 bones was recovered, most of it representing kitchen refuse, giving a remarkable record of the wild and domestic animals used as food. Over 97 percent of the bone recoveries were from domestic animals indicating that hunting had only minor importance in food provision. The domestic species represented were cattle, sheep and goats, pigs, horses, donkeys, cats, dogs, chickens, geese, and pigeons. Cattle were by far the most prevalent in bone recoveries (38 percent), followed by sheep and goats (21 percent) and pigs (19 percent).

Chickens appeared to have an important role in food economy of the town (Bökönyi, 1984). They represented four percent of the total bone recoveries. Nearly all were tarsometatarsal bones and they could be sexed based on presence or absence of a spur. Some of the large tarsometatarsi had only a very rudimentary spur and these were considered to be capons, which are known from literary record to have been produced by both Greeks and Romans. No doubt this interpretation is incorrect because spurs continue to grow in males that have been caponized. It is more likely that those with rudimentary spurs were sexually immature. Reassessment of these specimens is warranted. Plotting of bone measurements clearly indicated presence of at least two types of birds. The smallest, estimated to weigh 1.0-1.5 kg, were thought to be local unimproved stock, about the size of junglefowl, which had been introduced to Hungary by the Scythians. The larger type, estimated to exceed 2 kg, were believed to be improved Roman stock. Chicken bones similar in size to these have been found at all other Roman sites excavated to date. The sex ratio among bone recoveries was 79 hens : 39 roosters : 25 capons (?). All of them were adults. Very few subadult skeletal remains were found. Bökönyi (1984) speculated that the high proportion of males among the bone recoveries may have resulted from the Roman practices of slaughtering only adult poultry and of keeping many males for breeding. He also speculated about use of males for cock-fighting but there was no evidence from the excavations to indicate that the sport was practiced.

Europe. Introduction of chickens to Europe north of the Mediterranean is poorly understood. According to Brown (1929), chickens reached Europe by two routes. They came via Iran and the Mediterranean, as documented both in archaeology and in literature. And he believed they also travelled by a northern route across China and Russia arriving about A.D. 1300. West and Zhou (1988) have proposed a similar northern routing inferring that these birds derived from a Chinese domestication and not from the Indus valley domestic stock. Peters (1913) also proposed two routes but both were from Iran, and thus from the Indus valley; one was via the Aegean Sea to the Greeks and Romans, and the other was via the Black Sea and Scythia reaching central Europe through southern Russia. Bökönyi (1984) stated that chickens were first brought to Hungary by the Scythians whose empire flourished about 500 B.C., thus favoring Peters' second route. Carter (1971) has also provided support for Peters' two routes based on linguistic evidence. He demonstrated gradual change of the Sanskrit world *pil* from India to the Mediterranean and western Europe where it became *pullet* and *gallus*, and evolution of Sanskrit *kukuta* through central and northern Europe to become *chicken* and *cock*.

The summary of archaeological evidence prepared by West and Zhou (1988) indicates that the main period for dispersion through Europe was the Iron Age, but chickens were already present in some areas during the late Neolithic and early Bronze Age. Chickens were widespread in Europe by the first century B.C. (Zeuner, 1963). Bones dating from this period have been found in Switzerland, Germany, and England. Caesar in *De Bello Gallico* specifically mentioned that the Celts of Britain had chickens before the Roman conquest, but they would not eat them. Bate (1934) described the anterior half of a chicken sternum found at Colchester that dated to A.D. 43 at the latest and hence was pre-Roman. Zeuner (1963) mentioned another specimen from Wiltshire which may have lived just before the Romans arrived or just after; its identity as a chicken has been confirmed by Harrison (1978). The Romans certainly brought their own chickens with them and bones have been recovered from a number of Roman sites (Zeuner, 1963). An unusual skull has been found at a Romano-British temple complex in Somerset dating to about A.D. 350 (Brothwell, 1979); it exhibits frontal expansion as a result of cerebral hernia and probably was a crested bird as seen now in the Polish breed. Claudius Aelianus, a Roman writer of the second century A.D., described such crested chickens (Petrov, 1962).

The Romans are credited with popularizing chickens throughout their empire and secondarily with their spread beyond Roman boundaries, although chickens were known earlier across most of Europe. Hehn (1885) summarized linguistic similarities in chicken terminology among European nations, giving clues to origins but no conclusive evidence. Zeuner (1963) described sacrificial use of chickens both within and beyond the Roman sphere of influence. And Petrov (1962) briefly described archaeological evidence for chickens in southern Russia during this period.

With decline of the Roman Empire, poultry keeping seemed to have diminished in importance throughout Europe, and chickens became farmyard scavengers (Wood-Gush, 1959) not regaining importance until the beginning of the 19th century. Historians have paid almost no attention to the role of chickens during this interval, except for the recent work of Stephenson (1987) concerning poultry keeping in medieval England and that of Requate (1960) cited by Zeuner (1963) concerning central and northern Europe.

Diffusion of chickens in Africa. Except for Egypt, little is known about introduction of chickens to Africa. Sauer (1969) said they were already present at the time of first European contact. Chickens with black feathers, meat, and bones were described from Mozambique in 1635. The fibromelanosis mutant was known in India at that time but not in Europe. Carter (1971) showed that words for chickens in both East and West Africa had an Indian root. Origin from India is likely because trade between India and the east coast of Africa was well-developed at an early date.

Diffusion in southeast Asia and the Pacific islands. Spread of chickens eastward from the Indus valley has received little study. Indeed, if it is shown that a separate and earlier domestication had occurred in China, or if it is shown conclusively that chickens were first domesticated somewhere in southeast Asia (Darwin, 1896; Peters, 1913; West and Zhou, 1988), then a reassessment and reappraisal of diffusion will be needed. According to Carter (1971), names for chickens in China and in the Philippines are similar, tracing to the Zhou dynasty, 1122-249 B.C. Japanese words are totally different, as are their chickens, probably indicating a different origin. West and Zhou (1988) suggested that Japanese chickens may have been acquired from China via Korea during the Yayoi period (300 B.C. - A.D. 300) based on bone discoveries from that period on Iki Island. Evidence based on both morphology and linguistics suggests introduction to the Pacific islands from both China and India. The excellent monograph by Ball (1933) on junglefowl of the Pacific islands contains a wealth of historical and biological information that would be helpful in a reassessment of origins and dispersal.

Pre-Columbian chickens in America. The possibility of chickens being present in the Americas in pre-Columbian times continues to interest agricultural historians. The usual view is that chickens first arrived during the Spanish Conquest and spread rapidly thereafter through South and Central America. Much later the English, French, and Dutch brought them to eastern North America. But there is a strong minority opinion to the effect that chickens arrived in Central and South America long before the Spanish Conquest, and that they came from across the Pacific.

Jett (1983) had provided an overview of the evidence for pre-Columbian transoceanic contacts. The earliest great sailing tradition was the southeast Asian sailing raft, followed by the Indonesian and Oceanian sailing canoes, and the eastern Asian junk. All of these date from before the Christian era and were far superior to any contemporary watercraft known in the West. It has been demonstrated repeatedly that these craft were capable of very prolonged ocean voyages; they could easily have reached the Americas by crossing the Pacific, and they could easily have returned. Jett (1983) described the major seagoing cultures of the eastern Pacific Rim which might have crossed the Pacific - Japanese, Chinese, Malaysian, and the Hindu-Buddhist kingdoms of India and southeast Asia. He related these to contemporary high cultures of the Americas - particularly the Olmec of Mexico, the Chavin of Peru, the Mayan of Yucatan, and the Aztec of Mexico. There is no conclusive proof of trans-Pacific contact, but the cumulative evidence is very strong. There are some startling observations. In Peru there are 95 place names that have no meaning in local languages but they have a meaning in Chinese, and 130 place names that correspond to Chinese place names. Coastal natives of British Columbia had Japanese slaves at the time of first European contact. The peanut is native to South America and was domesticated there, but the Chinese had it about 3000 B.C. And India may have had maize (corn) by A.D. 1100. Kinds of dogs, and their uses, were similar in South America and Asia. Very strong similarities between Asian and American cultures can be found in architecture, art and decoration, tools and weapons, religious practices, and language. The list is nearly endless. But absolute proof of pre-Columbian contacts and general acceptance by scholars remains elusive.

Carter (1971), and more recently Johannessen (1982), have focused their attention on whether chickens might have been acquired by the Americas through pre-Columbian transoceanic contacts. The subject has also received attention from Wood-Gush (1959) and Sauer (1969). A massive amount of biological, cultural, and linguistic evidence was reviewed and interpreted by Carter (1971); the evidence is cumulative and strongly suggests introduction of chickens to South America from across the Pacific many centuries before the Spanish Conquest. Conclusive proof would be provided by skeletal remains from pre-Columbian sites, but thus far none has been documented in the literature, although Carter (personal communication, 1988) insists that such discoveries have been made.

Distribution of chickens within South America early in the 16th century is puzzling. The Spanish certainly brought chickens with them, calling them Castilian hens. The Spanish and Portugese explored most of South America during the 40 years following first discovery, and everywhere they went they found chickens, presuming them to be descendants of the first Spanish introductions. The rate of spread, estimated by Carter (1971) at 80-160 km per year, seems exceedingly rapid. His estimate of diffusion rate in Europe and Asia was 1.5-3 km per year which is comparable to rate of spread for other new things. He believed instead that introduction and spread of chickens in the Americas may have paralleled and been contemporary with that of maize (corn) which began about 1500 B.C. Rate of diffusion would then be similar to that calculated for chickens in Europe and Asia.

The linguistic evidence for pre-Columbian introduction cited by Carter (1971) is that names used in historic times were Spanish in those areas where it is known that chickens were introduced directly from Spain, and they were non-Spanish in areas remote from Spanish influence. He noted similarity in some names used in the Americas and those used in Asia.

Cultural use of chickens also indicated dual origin. In tropical forested South America chickens were not used for food, in keeping with Asiatic practice, but they had considerable importance in religious and cultural practices. Under Spanish rule, food use was very important. Although Carter (1971) devoted considerable attention to cock-fighting, he later stated that there was no trace of cock-fighting in pre-Columbian America so that introduction may have preceded development of the sport in Asia (Carter, 1975). Cock-fighting certainly became popular after Spanish and Portugese colonization; the Chilean Finsterbusch (1929) wrote a detailed historical account of the sport throughout the world and especially in Latin America.

Johannessen (1982) has made a detailed study of fibromelanotic chickens in present-day culture of the conservative K'ekchí Indians of Guatemala and has drawn a comparison with cultural use of the same mutant in China. He described them as black-boned black-meated chickens. Those of the K'ekchí-Maya can be any feather color although black is preferred, they often have pea or walnut comb, and they may be crested and booted. He described some as having tufts of feathers extending laterally at the level of the ears and protruding 1-2 cm from the side of the head; it is not clear whether this description refers to the ear-tuft mutation or to muffs and beard. Silky feathering is not found among them. There is a remarkable similarity between present-day K'ekchí and both ancient and modern Chinese use of the birds in medicinal cures for both physical and psychological illnesses and in witchcraft. Neither culture will eat the birds, although chicken eggs and meat from non-fibromelanotics are readily consumed. Johannessen's (1982) conclusion is that the K'ekchí birds and their use were derived long ago from China.

Carter's (1971) biological data pertain mostly to whether American chickens were Asiatic or Mediterranean in type, and to the morphological and color variants that occurred among them. A detailed assessment of his evidence was made by Crawford (1984). Not all of Carter's genetic interpretations were correct, but he was able to demonstrate that chickens in areas away from Spanish influence had Asiatic traits and were quite different from Castilian chickens, and he was probably correct in assuming that most of the single gene variants were Asiatic in origin. However, there was very active seagoing trade between southeast Asia, Africa and South America involving Spanish, Portugese, and Dutch soon after the Spanish Conquest; Asiatic chickens could easily have been introduced in that way. Indeed Finsterbusch (1929) claimed this origin for much of the South American cock-fighting stock. Nearly all of the mutants claimed to be Asiatic were also known in the Mediterranean area and in Africa at the time - pea comb, silky, frizzle, rumplessness, fibromelanosis, crest - although they were not necessarily common there. Aldrovandi described them all from Italy in A.D. 1600 (Lind, 1963). However it may be significant that other mutants which were prevalent then in Europe - polydactyly, rose and duplex comb, white egg-shell - did not appear to exist in the early American stocks.

The claim for pre-Columbian origin of American chickens has been clouded by presence of blue egg and ear-tuft traits. Neither trait is known among Asiatic chicken stocks. The ear-tuft trait was first reported in 1921 by Castello (1924), and genetics of it was only recently deciphered (Somes, 1978). It is very different from the muffs and beard condition, and is known only in the Araucana chickens from South America. Castello (1924) drew first attention of the scientific community to the blue egg trait, and its inheritance was described by Punnett (1933). It must be of considerable antiquity since it was known in many parts of South America, Mexico, and Central America. Occasional fragments of evidence indicate that the blue egg condition may also have arisen independently in northwestern Europe within the last several centuries, perhaps in Scandinavia or perhaps on islands off the north and west coasts of Scotland (Crawford, unpublished; Hutt, 1988, personal communication). It seems peculiar that Darwin (1896) made no mention of either blue egg or ear-tuft. He journeyed extensively overland in South America during his tour as biologist with the *Beagle* expedition. Since his main interest was variation in animals and plants, and since he studied and wrote about poultry, it is unlikely that he encountered or heard about these two variants. Except for their occurrence only in South American chickens, and except for the widespread occurrence of the blue egg trait, the two mutants add little to the debate over chicken origins.

Overall, genetic evidence favoring pre-Columbian introduction of chickens to the Americas is weak. But when combined with Carter's (1971) linguistic and cultural evidence it becomes more convincing.

There are many who oppose the view that chickens were present in the Americas in pre-Columbian times. Most of their writings have been reviewed and criticized by Carter (1971). An important paper which seems to have been overlooked is that of Termer (1951). He was particularly interested in the domestic livestock and poultry of Mexico and Central America around the time of the Spanish Conquest. He examined letters and documents written by the explorers and their retinues, and concluded that there were no chickens there until the Spanish brought them. He believed that conflicting evidence could all be traced to faulty translation and transliteration of words. He drew attention to the establishment of livestock breeding farms, which included Spanish chickens, in Cuba and Santo Domingo as early as 1495. These served as important supply depots for expeditions in the following decades, including the conquest of Mexico by Cortez which began in 1519. Termer (1951) believed that chickens were adopted very rapidly by native peoples, not as food, but as a trade item in preference to the more expensive turkey.

A chicken bone recovery from a known pre-Columbian site would close the debate.

TURKEY

Domestic turkeys (*Meleagris gallopavo*) were found in Mexico by the Spanish Conquistadores and were taken immediately to Europe. They had been derived from the Mexican subspecies *M. g. gallopavo*. They were brought to eastern North America from Europe beginning in the 16th century, where they interbred with the eastern subspecies *M. g. silvestris* forming the bronze bird that became the foundation for nearly all domestic stocks. A separate domestication of *M. g. merriami* took place in the southwestern United States but there is no evidence that it has persisted or that it contributed to modern domestic turkeys.

Wild Species

Classification. Turkeys are classified as order Galliformes, suborder Galli, family Phasianidae, subfamily Meleagridinae, and genus *Meleagris* (Howard and Moore, 1984). Two distinct species are now recognized; formerly the two were placed in separate genera. The species from which domestic turkeys descended is the true wild turkey *M. gallopavo* Linnaeus 1758. Seven subspecies are recognized based mostly on geographic range and plumage differences (Howard and Moore, 1984; Porter, 1985); these are *M. g. gallopavo* (Mexican), *intermedia* (Rio Grande), *merriami* (Merriam's) *mexicana* (Gould's), *silvestris* (eastern), *oneusta* (Moore's), and *osceola* (Florida). The ocellated turkey (*M. ocellata*) was formerly placed in a separate genus *Agriocharis*. It has never been domesticated. Hybrids between wild turkey and ocellated turkey can be obtained but success is poor (Gray, 1958). It is very unlikely that ocellated turkeys have made any contribution to the domestic form. There appear to have been no planned studies of hybrids among the subspecies of *M. gallopavo* or with domestic turkeys, but it is unlikely that serious reproductive barriers would be found.

Description. Wild turkeys are very much like the barnyard bronze domestic birds, except for much slimmer build. They do not have the massive muscling of breast and thighs found in modern domestic turkeys. According to Aldrich (1967), the eastern subspecies have brown tips on the tail feathers and upper tail coverts, and a bronze tone to the entire plumage as seen in domestic bronze turkeys. Southwestern forms have pale tips on the tail feathers and upper tail coverts leading to their designation as white-rumped, and the entire plumage has a metallic green tone so that their feathers appear to be black; presumably this was the coloring of the first domesticates to reach Europe from Mexico. There are two molts per year, a partial prenuptial molt in late winter and a complete molt in late summer; there is no flightless period (Porter, 1985). Long (1981) gave body weights of 4.7-15.8 kg in males, and 2.1-6.3 kg in females. Porter (1985) summarized biology and ecology of wild turkeys stating that a very broad range of habitats can be used, but preference is for mature forest interspersed with open fields. They are omnivorous and essentially non-migratory. Sexual maturity can be reached in the first year under good environmental conditions. Clutch size is about 11 eggs, the eggs weighing 65 g. Incubation time is 26-28 days and hatching success can exceed 90 percent.

Distribution. Wild turkeys formerly occupied a very large range in North America, including Mexico from the Rio Balsas valley northward, most of the United States east of the Rocky Mountains, and southern Ontario in Canada (Aldrich, 1967). They were exterminated in many areas soon after first settlement through hunting pressure and habitat destruction. Both Leopold (1959) and Aldrich (1967) commented on the rarity of the Mexican subspecies in its native range in recent times. Many successful reintroductions have been made (Long, 1981), but care has not always been taken to maintain subspecies identity during captive breeding and to utilize subspecies originally native to an area, so that it may no longer be possible to find pure populations of a subspecies in the wild. Schorger (1966) suspected that most populations east of the Mississippi now contain genes from domestic birds.

Wild turkeys have been successfully released as game birds in Canada far beyond their normal range, in the Hawaiian Islands, Germany, New Zealand, and on Prince Seal Island near Tasmania (Long, 1981). Other introductions did not persist.

Domestication And Early History

Mexico and Central America. The most comprehensive accounts of turkey domestication and early history in the Americas are contained in a paper and a monograph by Schorger (1963, 1966). The story was summarized in Mason's *Evolution of Domesticated Animals* (Crawford, 1984) and it is repeated here in more detail.

Place and date of domestication in Mexico are not known with any certainty. Schorger (1966) described the finding of bones from domestic birds at an archaeological site in the valley of Tehuacán in present-day Puebla state which dated in the Palo Blanco period, 200 B.C. - A.D. 700. Indirect evidence of domestication early in that period was provided by finding *M. gallopavo* bones at a Guatemalan archaeological site dated about A.D. 700; since turkeys were not native to Guatemala, they must have been brought from the north where they presumably had been domesticated much earlier (Schorger, 1963). The domesticated birds found by the Spaniards were only about half the size of wild turkeys and there were color variants among them (Schorger, 1963, 1966), both phenomena indicative of long domestication. Sahagún described the color variations in a manuscript completed about 1570; Schorger's (1966) translation of the passage indicates presence of self-colors rather than variations within a plumage: "... (the birds) ... were of various colors; some white, others red, others black, and others brown". White turkeys are mentioned frequently in writings from the time of the Spanish Conquest. Almost all early records of turkeys refer to their use as meat, both by the Spanish and by the native peoples. Schorger (1963) cites only one exception in which turkeys were kept by certain Tarascans of Michoacan for feathers to decorate their gods and not for food. Because the Aztecs and Tarascans achieved the highest development of turkey culture, Leopold (1959) speculated that domestication occurred within their territories in the western highlands of Mexico, perhaps in Michoacan. However, Schorger (1963, 1966) made no speculation about precise time and precise place of domestication.

Distribution of domestic turkeys in Mexico and Central America at the time of the Spanish Conquest is known reasonably well. Schorger (1963) gave a very detailed review from writings of that time, including in many cases the numbers of turkeys to be paid in tribute to Aztec rulers and Spanish conquerors. When the Spanish arrived, they found domesticated turkeys throughout Mexico southward from about latitude 24° N. None were recorded from Sonora in the north until 1763, and there are no early records for Chihuahua and the northeastern states. All archaeological findings of turkey bones in the Yucatan Peninsula have been shown to belong to the ocellated turkey which is native there, indicating that the Mayas did not have domestic turkeys until shortly before the Conquest. The Spanish recorded their presence there as early as 1517. Domestic turkeys were recorded by explorers as far south as the Nicoya Peninsula in Costa Rica, which seems to have been the limit of their pre-Columbian distribution. A pre-Columbian turkey effigy jar was found there confirming the written evidence; it was located during archaeological search of an outpost colony of Mexicans who probably had brought domestic birds with them.

Although most evidence indicates that the domesticated turkey of Mexico was derived from *M. g. gallopavo*, the possibility of contribution to the domestic bird from other wild subspecies should not be overlooked. Schorger's (1963, 1966) map of areas where turkeys were being raised in the 16th century includes natural territory of Gould's (*M. g. mexicana*) and Rio Grande (*M. g. intermedia*) subspecies. The Rio Grande turkey was said to have been domesticated in one locality, and Gould's turkey was said to be easily tamed, but it is not known whether these persisted as domestic stocks. Merriam's turkey (*M. g. merriami*) was certainly domesticated independently in southwestern United States.

The Caribbean and South America. There is no record of pre-Columbian turkeys on islands of the Caribbean, but they must have been introduced very early by the Spanish. Schorger (1966) recorded that they were taken to Cuba between 1511 and 1517. Shipments of turkeys to Spain in 1512 and to Rome in 1520 have been recorded from Hispaniola although that may have been only a trans-shipment base. Turkeys were known in all islands of the Antilles by 1667.

Diffusion of turkeys into South America is poorly documented. Schorger (1966) states that they were present in Peru before arrival of the Spanish and may have been taken there from Nicaragua. There are records of domestic turkeys in Ecuador by 1587 but they were not known in Chile until about 1646. It seems clear that if domestic turkeys were a pre-Columbian arrival in South America, they had only just arrived by the time of the Conquest.

First discovery and introduction to Europe. Precise dates for first discovery of domesticated turkeys by Europeans, and for first introduction to Europe, are conflicting (Schorger, 1966). Columbus may have been the first; he landed on the coast of Honduras on August 14, 1502 and was given *gallinas de la tierra* by the natives. Two years earlier Vincente Yáñez Pinzón was given what may have been turkeys at the Gulf of Paria in Venezuela, although that location was far beyond the known pre-Columbian range of domestic turkeys. And in 1499 Pedro Alonso Niño is recorded as discovering turkeys on the coast of Cumana, Tierra Firme (at that time comprising Panama, Costa Rica, and Nicaragua) and he is said to have taken them to Europe in 1500. Precisely dated records have been found in Spain which refer to European introductions in 1511 and 1512. One document dated 24 October 1511 was an order from the Bishop of Valencia for each ship from the Islands and Tierra Firme to bring to Seville ten turkeys, half males and half females, for breeding. Another document dated 30 September 1512 from the King refers to two turkeys which had arrived in Spain from Hispaniola. Cortés began the conquest of Mexico in 1519 and thereafter domestic turkeys rapidly became known and presumably shipment to Europe became a frequent event.

Europe. Diffusion of turkeys through Europe was very rapid. Following recorded introduction in the first decades of the 16th century, they were known throughout Europe by mid-century. Schorger's (1966) review includes the following first records of turkeys: Italy about 1520 when a pair including a white female was sent from Hispaniola to Rome, France by 1538 and well-known by 1552, Norway and Denmark about 1550, Sweden in an inventory of a King's farm by 1552 and on private farms by 1600, Germany at a wedding feast in 1560. Moubray (1854) stated that an English family has a tradition that one of their ancestors brought the first turkeys to England in 1521. In 1550, in recognition of this tradition, William Strickland of Yorkshire was granted arms and a crest consisting of *A Turkey Cock Argent beaked and legged Sable combed and wattled Gules*; the drawing which is part of the grant docket is unmistakably that of a turkey (Brooke-Little, 1985). But it has not been resolved whether the Strickland turkey was a domestic bird from Mexico or whether it was a wild turkey from eastern North America. The latter is more likely since William Strickland is said to have accompanied Sebastian Cabot on some of his voyages to the New World (Schorger, 1966). The first accepted record for domestic turkeys in England is 1541 (Schorger, 1966) when an archbishop decreed that only one each of turkeys, swans, and cranes should be served at any entertainment because they were such rare delicacies (Moubray, 1854). By 1555 they were plentiful.

The speed with which turkeys became known in Europe has not been adequately explained. Crawford (1984) speculated that massive numbers might have been shipped from the New World to the Old at the time of the Conquest, because normal reproduction of the few known to be imported would be inadequate for the rapid and widespread diffusion that occurred through Europe and elsewhere.

Others have speculated whether turkeys had already reached Europe by the time of the Spanish Conquest, perhaps crossing the Pacific and entering Europe from Asia. Carter (personal communication 1975, 1987) has linguistic evidence to indicate introduction to Europe from Asia but cannot document a known pre-Columbian turkey bone in either continent. Bökönyi and Janossy (1958) described a tarsometatarsus found in excavating a 14th century stratum of the Royal Castle of Buda in Hungary, and cited a similar bone discovery (late 13th century) from ruins of the Castle of Schwaniburg, in Switzerland. They thought that their bone was from a turkey, but Schorger (1966) had it re-examined and determined that it was from a peafowl. Similarly, two signet rings from Hungarian graves of A.D. 900-1200 were thought to bear images of turkeys (Bökönyi and Janossy, 1958). However, based on sketches of the rings, one is more likely to represent a peafowl, and the other only faintly resembles either turkey or peafowl (Crawford, 1984). And finally, there was a controversy concerning turkeys depicted on the frieze of a mural in Schleswig Cathedral (Schorger, 1966; Bökönyi and Janossy, 1958). The mural was supposedly painted in 1280 and the presence of turkeys in it was taken as evidence that the Norse may have brought turkeys to Europe from North America. But it is believed now that turkeys were not part of the original mural, being added by a restoring artist when the mural underwent cleaning and repair in 1890-1891. Thus far, no compelling evidence has been advanced to support a belief in entry of turkeys to Europe through Asia. Introduction directly from Mexico and Central America in Columbian times is the more tenable hypothesis.

Eastern North America. An essential part of the history of domestic turkeys concerns introgression of the eastern wild turkey (*M. g. silvestris*) into the domesticated Mexican bird. The Europeans who colonized eastern North America beginning in the 16th century included turkeys among their settler effects. Schorger's (1966) review indicates presence of domestic turkeys in Virginia in 1607, Massachusetts in 1629, and Quebec in 1647; they were plentiful in Pennsylvania and New Jersey by 1698. American poultry writings, especially those from late 19th and early 20th centuries, contain frequent discussion of turkey breeding. It was quite usual for domestic turkey hens to be courted and bred by wild toms. The resulting crossbreds assumed the bronze plumage of the *silvestris* parent, losing the black appearance of the Mexican bird. They were much larger than the Mexican parent. When superior size and vigor became evident, crosses were purposely arranged using captive wild toms. These practices were very widespread, occurring from Ontario and Quebec to Florida, so that introgression from *M. g. silvestris* became a general phenomenon. The hybrid domestic population, called American Bronze, reached Europe early in the 19th century and had become vastly popular by the end of the century (Brown, 1929). Both in Europe and in North America it eventually evolved into the modern broad-breasted type.

The eastern wild turkey (*M. g. silvestris*) that played such an important role in this genetic event was never domesticated (Schorger, 1966). Natives and settlers frequently kept wild birds in captivity, but wildness was never lost and no instances of progress in domestication are known.

Independent domestication in southwestern United States. Although the story is not generally known, Merriam's turkey (*M. g. merriami*) was domesticated by the agricultural Pueblo people of the southwestern United States about the same time as domestication of turkeys was in progress in Mexico. Aldrich (1967) and Schorger (1966) both indicate that the two events were independent and not borrowed one from the other, because of the geographic distance between the two cultures. There is no evidence that the Pueblo stocks contributed to modern commercial turkeys.

There is a large literature on archaeology and anthropology of the native cultures in southwestern United States. It contains scattered references to turkeys which have been summarized by Schorger (1966). The cultures were primarily agricultural in an arid environment. Settlements were built in caves and on mesas for defence, and access was purposely difficult. Evidence indicates that turkeys were kept in total confinement within the settlement houses, or they may have been free to roam within a village but not outside its walls. They were being kept about A.D. 500-700 and may have become domesticated then. There is no doubt about domestication by A.D. 700-900. Archaeological remains have been found in Nevada, Utah, Colorado, Arizona, and New Mexico. Coronado found domestic turkeys in New Mexico during his journey of 1540-1542, and the Rodriguez expedition found them in Dona Ana county of New Mexico in 1581. There is little evidence that the birds were kept as food, but abundant evidence for use of feathers, spurs, and bones for ornament, for weapons, and as tools. Lack of use as a food probably indicates an early stage of domestication. There is no indication in the literature that feather color mutants had been present. Schorger (1966) noted that keeping of turkeys had declined by the 19th century. It is not known whether turkey culture ceased then or whether it persists among Pueblo peoples to the present.

JAPANESE QUAIL

All or nearly all modern domestic Japanese quail were derived from the wild Japanese quail, which is now recognized as a distinct species *Coturnix japonica*. Domestication began about the 11th century in Japan, China, or Korea. Independent domestication of the closely related common quail (*Coturnix coturnix*) may have occurred elsewhere but strong evidence is lacking, and there is no certainty that such stocks have persisted to the present.

Wild Species

Classification. Confusion and controversy have persisted in names for the modern domesticated form of *Coturnix*. The usual scientific nomenclature has been *Coturnix coturnix japonica* and there has been a variety of vernacular names in everyday use. It seems clear that most, if not all, modern domesticated *Coturnix* have been derived from the wild Japanese form. Taxonomists now agree that the wild Japanese quail represents a species distinct from the common quail (*Coturnix coturnix*) of Europe, Asia, and Africa, and it should no longer be regarded as a subspecies. Hence, throughout this treatise the popular and scientific designations that will be used are Japanese quail and *Coturnix japonica.*

According to the American Ornithologists' Union (1983), and Howard and Moore (1984), Japanese quail belong to order Galliformes, family Phasianidae, subfamily Phasianinae, genus *Coturnix*. Within genus *Coturnix* there are five recognized species (Howard and Moore, 1984). These are: common quail (*C. coturnix*) comprising six subspecies widely distributed through Europe, Africa, and Asia; Japanese quail (*C. japonica*) from Mongolia, Sakhalin and Japan; black-breasted quail (*C. coromandelica*) from India, Sri Lanka, and Burma; harlequin quail (*C. delgorguei*) comprising three subspecies from Africa and Arabia; and pectoral quail (*C. pectoralis*) from Australia. The American Ornithologists' Union (1983) also gives separate species status to *C. japonica* Temminck and Schlegel, and combines it with common quail (*C. coturnix*) in a superspecies. The two are distinguished as full species because of differences in vocalizations and sympatric breeding in northern Mongolia. According to Baker and Manwell (1967), the two do not hybridize readily and any progeny are sterile or have low fertility.

Description. There are several major treatises on natural history and biology of *Coturnix* which include information on *C. japonica* (Fitzgerald, 1969; Toschi, 1959; Wetherbee, 1961). These were written before separate species status for the Japanese form had been accepted, and hence descriptions and discussions do not always clearly distinguish between common and Japanese quail. The treatise by Taka-Tsukasa (1935) deals exclusively with the Japanese species.

Wetherbee (1961) described birds of the genus as small, chubby, tailless, cinammon-colored, terrestrial galliforms, that are extremely variable and adaptable in their biology. Except for smaller body size, wild Japanese quail are very similar to the domestic form (Kawahara, 1967, 1973). Body weights of wild adults were found to be 98 g for males and 102 g for females; shank lengths were similarly about 20 percent less than in domestic birds. Wild-type plumage has been retained in domestic Japanese quail.

Preferred habitat is seeded pastures and meadows, from coastal plains to highlands up to 1000 m above sea-level (Kawahara, 1967). The birds live in pairs during the breeding season but during migration and in the winter they gather in large flocks. Their diet is omnivorous.

As might be expected, reproductive activity is much reduced in wild birds compared with domestic stocks. Kawahara (1967) reported that wild birds in Japan laid 7-12 eggs per nest. Only the female incubated the eggs and cared for the young. Incubation time was found to range from 16-21 days. Consult Taka-Tsukasa (1935) for details on natural history and breeding.

Distribution. According to Wakasugi (1984), wild Japanese quail breed across Mongolia, Manchuria, and Korea, and on the islands of Sakhalin, Hokkaido, and Honshu. Their northern limit is about 55°N. They are sympatric in Mongolia with *C. coturnix*. Most populations migrate southward for the winter to southern Honshu, the Korean peninsula, Taiwan, Hainan, and eastern China. Their southern limit is about 17°N.

Many introductions of *Coturnix* have been made (Long, 1981; Wetherbee, 1961). The only introductions having permanent success (Long, 1981) have been those to Réunion in the Indian Ocean (*C. coturnix africana*) and to the Hawaiian Islands in the Pacific Ocean (*C. japonica*). The Hawaiian introductions to Maui and Lanai in 1921 may hold special genetic interest; if the introduced birds were domestic or semidomestic they may be a feral remnant of the early stocks bred by the Japanese for song, which were extinguished during World War II (Cheng, 1988, personal communication).

According to Wetherbee (1961) introductions to North America occurred in three stages. Many thousands of *C. coturnix coturnix* from Sicily were released into northeastern United States between 1877 and 1882 but they did not become established. Secondly, huge numbers of 'Migratory Chinese Quail', believed now to be *C. japonica*, were imported by restaurants on the west coast of the United States from 1895 to 1904; some of these escaped or were released but none survived. Then in the 1950s, game-farm propagated quail were released in large numbers in southeastern and southcentral states, being derived from stock imported from Japan in 1953 (*C. japonica*); these introductions too were a failure. There have been many hopeful and well-intentioned releases since then in North America, all without lasting success, so that no further large-scale attempts to establish wild or feral populations of *Coturnix* in North America are being made. However the birds of the 1950s have survived and become popular in laboratory colonies and in commercial farms for egg and meat production, especially in North America but also in other countries.

Domestication And Early History

Very little information is available concerning domestication and early history of Japanese quail. The record on possible early domestication of common quail is similarly wanting. Acceptance of *C. japonica* as the sole ancestor of modern domestic Japanese quail may encourage scholars to seek further into its history. There is also a need to document the development of domestic stocks of *C. coturnix* if in fact they have ever existed.

Wetherbee (1961) advanced the claim that *Coturnix* was the first bird domesticated by man, but he provided no evidence. Zeuner (1963) mentioned quail only very briefly. He illustrated a hieroglyph from Old Kingdom Egypt (about 2500 B.C.) which almost certainly represents a quail and not a chicken, there being no record of chickens in Egypt until about 1840 B.C., but proof of domestication is lacking. Quail were also known to the Greeks and Romans who kept them for fighting but not for food. Presumably both authors were referring to common quail (*C. coturnix*), but it is not clear whether they were referring to captive wild birds or to incipient domestic stocks.

The history of domestic Japanese quail is only fragmentary. Domestication occurred in about the 11th century, perhaps in Japan, or they may have been introduced there as domestic birds from China or Korea (Howes, 1964; Wakasugi, 1984). The first written records in Japan date from the 12th century (Howes, 1964).

The birds were first kept and bred for song, especially by the Samurai (warrior) caste. Taka-Tsukasa (1935) has given a detailed account of the breeding and care of song quail and of singing competitions. He stated that song quail were particularly popular during the years of Keicho (1596-1614) and Kan-ei (1624-1643), and again during the years of Meiwa and An-ei (1764-1780); there was a brief revival of interest in 1918-1920. All of the song quail in Japan became extinct during World War II (Howes, 1964; Wakasugi, 1984). Howes (1964) made brief reference to independent selection of quail in central Europe for singing ability, indicating only that popularity also declined during World War II; more information would be desirable, particularly on whether these European song quail were *C. coturnix* or *C. japonica*.

The Japanese became interested in improving egg and meat production of their birds beginning about 1910 and continuing to 1941, and during Japanese imperial expansion at that time the improved stocks became established in Korea, China, and Taiwan (Howes, 1964).

It is believed that only a few domestic birds survived World War II in Japan (Howes, 1964; Wakasugi, 1984). They were used to rebuild the quail industry. It has been speculated that some birds may have been returned from Korea, China, and Taiwan to help in the rebuilding process, and that wild Japanese quail may have been added to the domestic stocks (Howes, 1964). Recovery was very rapid so that by the 1950s the Japanese food industry was very strong and birds were being introduced to North America, Europe, and the Middle and Near East (Wakasugi, 1984).

The Americans were propagating Japanese quail in large numbers during the 1950s for release as game birds (Wetherbee, 1961), but concurrently they were also developing an interest in using the birds for egg and meat production and as laboratory animals. The pioneer report on use as a laboratory animal is that of Padgett and Ivey (1959). Within a decade of that report research colonies had been established in many laboratories throughout North America and elsewhere, and a research newsletter *Quail Quarterly* was being published. All indications are that both production and laboratory birds were derived exclusively from Japanese imports beginning in the 1950s. Similarly the domestic stocks in other countries probably all trace either to imports from North America or to imports directly from Japan. It remains possible that some stocks of domestic *Coturnix* in Europe and Asia may have an origin involving *C. coturnix* but such has not been recorded in the technical literature. Hence it seems safe to assume that all modern domestic stocks of *Coturnix* are descended from the wild Japanese quail (*C. japonica*).

GUINEA FOWL

All authors agree that the domestic guinea fowl was derived from the helmeted guinea fowl (*Numida meleagris*) of Africa. There were at least several independent domestications involving more than one subspecies. Present-day commercials stocks were probably all derived from the West African subspecies *N. m. galeata*.

Wild Species

Classification. Belshaw (1985) has classified guinea fowl as order Galliformes, family Numididae, but Howard and Moore (1984) placed them in family Phasianidae and subfamily Numidinae. There are four genera (*Agelastes, Guttera, Numida, Acryllium*) comprising seven species. Genus *Numida* consists of a single polytypic species *meleagris* and 22 subspecies. Crowe (1985) has used a simpler classification of *Numida meleagris* involving nine well-marked subspecies which fall into three groupings:

West African	- *N. m. galeata* and *sabyi*
East African	- *N. m. meleagris* and *somaliensis*
Central-South African	- *N. m. reichenowi, mitrata, marungensis, papillosa,* and *coronata.*

Description. Crowe (1985) described helmeted guinea fowl as opportunistic omnivores which inhabit open savannah and mixed savannah-bush. They are gregarious in the nonbreeding season, and monogamous as breeders. Females, especially in the breeding season, emit a characteristic two-note 'buck-wheat' call; males respond with a single note; both sexes have a rattling alarm call. Males are slightly larger than females but otherwise they exhibit almost no sexual dimorphism. Adult body size ranges from 0.7-2.0 kg (Long, 1981). The crown of the head carries a bony helmet with a horny sheath, and a pair of wattles hang from the gape. The nares are exposed, but in subspecies inhabiting hot dry areas the nares are surrounded with warts or cartilaginous bristles. Blood supply to the helmet, wattles, and cere may have importance in thermoregulation. The legs are long and powerful, lacking a spur. Plumage is monotypic. The ground color is black, with white spots intermeshed with white vermiculation; the spots on the outer margins of the secondaries are enlarged to form bars. Incubation time is 27-28 days, and clutch size varies from 6-10 eggs.

According to Crowe (1985), the West African *galeata* subspecies is small to medium-sized, and has a naked cere and rounded red wattles. *N. m. sabyi* is isolated in Morocco and differs very little from *N. m. galeata*. The East African *meleagris* and *somaliensis* subspecies are medium-sized, they have long bristles on the cere, and rounded blue wattles. The Central-South African group are relatively large birds. They have a naked cere (except for *N. m. papillosa* which has warts around the rim) and triangular-shaped blue wattles with red tips.

Distribution. Helmeted guinea fowl occur naturally throughout most of sub-Saharan Africa. There is an isolated northern population (*N. m. sabyi*) in Morocco (Crowe, 1985).

Many introductions have been made, some involving wild birds and some involving domestic stocks, and reintroductions have been made to areas of Africa where they had been exterminated (Long, 1981). The population in Yemen was probably introduced long ago; it is similar to the East African subspecies, for which some authors use the designation *ptilorhyncha*. The population in Malagasy was probably also introduced; it is classified as *N. m. mitrata*. Many oceanic islands have been stocked, not all of them successfully. Repeated introductions in New Zealand, Australia, and the United States met with general failure. There were introductions to most islands of the Caribbean, sometimes with wild birds and sometimes with domestic stocks which became feral. Some of these introductions were made in the 16th century and others arrived as live provisions on African slave ships. Populations flourished but many later became extinct because of hunting pressure and predation by the introduced mongoose. Viable wild or feral populations persist in Haiti, Dominican Republic, and Cuba.

Domestication And Early History

It is likely that many separate domestications have occurred in many separate places over time. According to Crowe (1985), wild populations of *Numida meleagris* readily become commensals of man, increasing in numbers and distribution because of the water, roosting, and feed resources resulting from human activity. However, unlike the situation for other poultry species, there is little indication in the historical record that guinea fowl were utilized other than as a human food resource. Belshaw (1985) makes only brief mention of their use in religion and folklore and of feathers in decoration. Eggs were probably of first importance and edible meat was secondary.

Information on history of domestication within Africa is scanty and, except for Egypt, depends on oral history. Belshaw (1985) stated that early domestication had occurred in two areas - in southern Sudan and in West Africa - but the dates are not certain. The process of domestication probably continues even now. Guinea fowl were depicted in a mural from the Egyptian fifth dynasty about 2400 B.C. but there is no evidence that the birds were domesticated then. They also appear in archaeological remains at Farnak dated from about 1900 B.C. and at Thebes (1570-1300 B.C.). It is supposed that they were artificially hatched and reared in large numbers concurrently with chickens during that period (Belshaw, 1985) but evidence is lacking; chickens are known to have been in Egypt at that time, but they were absent from the archaeological record in subsequent centuries, not appearing again until about 600 B.C. under Greek and Persian influence.

Guinea fowl were well-known to the Greeks and Romans in classical times. They were mentioned by Ovid, Aristotle, Pliny, Varro, and Columella. According to Zeuner (1963), the Greeks called the bird *melanargis* (black-white) which was corrupted to *meleagris* and became associated with Greek mythology. Moubray (1854) related that story: "... the Meleagrides, the sisters of Meleager ... who was cruelly put to death, bewailing the death of their unfortunate brother, were metamorphosed into Guinea-fowls, the shower of tears they shed bedecking their otherwise sable plumage with white spots ...". Wood-Gush (1985) indicated that the Moroccan subspecies *N. m. sabyi* was kept as a sacred bird on an Aegean island in the fourth century B.C. The Romans later knew this bird as the Numidian fowl. They also had birds of the East African subspecies which they called Meleagris and which they preferred. Both terms were eventually utilized by Linnaeus in formal naming of the genus and species. Guinea fowl were highly regarded as a food item by the Romans who must have distributed them throughout the Roman Empire. Zeuner (1963) mentioned bones of guinea fowl discovered in a Roman camp in the Taunus Mountains of West Germany, and Belshaw (1985) refers to a leg bone carrying a metal ring found in ruins of the Roman town of Silchester in England.

With decline of the Roman Empire, guinea fowl seem to have disappeared from Europe leaving almost no trace in the historical record. Mongin and Plouzeau (1984) refer to possible exceptions. They may have persisted in Greece and Italy, since Wood-Gush (1985) has noted a reference to the keeping of guinea fowl in Athens during the tenth century.

The Portugese of the late 16th century are generally credited with rediscovering guinea fowl on the west coast of Africa, from where the bird acquired its common name. The term *poule de Guinée* may have been used first in 1555 by Belon (Mongin and Plouzeau, 1984). The Portugese took these guinea fowl to Europe, to the Americas, and elsewhere. Diffusion through Europe was probably concurrent or perhaps slightly in advance of turkey introductions, resulting in unfortunate confusion of names and identity of the two species which persists in their scientific nomenclature.

Nearly all modern guinea fowl are likely to have been derived from Portugese introduction of the West African subspecies *N. m. galeata*. There are indications that new commercial hybrids may involve blends of several subspecies (Belshaw, 1985) but documentation in the technical literature is lacking. Domestic birds in Malagasy and those introduced from there to other localities may be domesticated *N. m. mitrata*. Those of eastern Africa are likely to be domesticates of *meleagris* and *somaliensis* subspecies, and those of the Mediterranean area may still bear traces of both East and West African subspecies.

RING-NECKED PHEASANT

After many centuries of captive propagation, ring-necked pheasants are still less than fully domesticated. A primary objective throughout has been to retain wildness in a captive bird being bred for sport shooting. Only in recent decades have some stocks been bred only for meat yield where tameness would be desirable. Domestic stocks were derived from the common pheasant *Phasianus colchicus*; subspecies from black-necked, white-winged, and grey-rumped groups have contributed. Some stocks also contain genes from the closely related green pheasant *P. versicolor*.

Wild Species

Classification. The classification scheme used by Beebe (1918-1922, 1936) has been followed by Delacour (1977), Howard and Moore (1984), and Johnsgard (1986) with only minor modifications. Pheasants are classified as order Galliformes, suborder Galli, family Phasianidae, subfamily Phasianinae, tribe Phasianini. The genus ancestral to domestic ring-necked pheasants is *Phasianus*, consisting of two species, common pheasant *P. colchicus* Linnaeus 1758 and green pheasant *P. versicolor* Vieillot 1825. The *colchicus* species exhibits continuous variation, especially in male plumage, across its very broad natural range. Thirty subspecies are recognized, and as a convenience they are combined into groups: black-necked 4 subspp., white-winged 6 subspp., Kirghiz 2 subspp., olive-rumped 1 subsp., and grey-rumped 17 subspp. *P. versicolor* of Japan is so closely related to the common pheasant that it could be considered to be an allospecies or a subspecies of *P. colchicus*. Three subspecies of *versicolor* are recognized.

Hybrids can occur among all species and subspecies of *Phasianus* (Delacour, 1977; Gray, 1958), and there appears to be no cause except geography for reproductive isolation. Zones of hybridization usually are found where ranges of subspecies adjoin. Most introduced and domestic stocks are believed to have a mixture of species and subspecies origins.

Description. Common pheasants are familiar birds in temperate climates throughout the world, but they are less well-known in warm and tropical areas. They have the general characteristics of Galliformes. They are relatively large and slender with a long tail, and males have erectile ear-tufts. Males are brilliantly colored and females are drab. There is a white neck-ring in eastern subspecies but not in western ones. The former predominate among introduced stocks in some countries leading to the popular name for domestic and semidomestic birds. Details of pigmentation in the numerous subspecies are described by Delacour (1977). In general the wild-type plumage pattern is retained in domestic stocks, but coloration is variable depending at least in part on which subspecies played a role in their development. Body weights range from 0.5-1.1 kg (Long, 1981).

Preferred habitat is patches of dense cover near cultivation, in steppe or in desert (Delacour, 1977). They tend to be sedentary, and appear as solitary individuals, in pairs, or in small groups (Long, 1981). As in most galliforms their diet is omnivorous.

They are polygamous and seasonal breeders. Clutch size is 8-14 eggs (Long, 1981), the eggs weighing 25-30 g (Delacour, 1977). Incubation time is generally considered to be 23 days.

Distribution. Natural range of pheasants is confined to suitable habitats in temperate areas of Asia (Delacour, 1977). *P. colchicus* extends across Asia from the Black Sea and Caspian Sea to Manchuria and Korea in a broad band between latitudes 35°N and 48°N. *P. versicolor* is found throughout Japan except on the island of Hokkaido.

Phasianus has been introduced widely throughout temperate parts of Europe and North America, and to suitable habitats in Hawaii, Australia, and New Zealand (Long, 1981). Probably no other avian species has been so widely exploited as a gamebird.

Domestication And Early History

According to legend, common pheasants were brought to Europe about 1300 B.C. by Jason and the Argonauts following their search for the golden fleece in Colchis along the Phasis river (Moubray, 1854). Pliny the Elder (A.D. 23-79) identified the same geographic origin, now known to be the Rion river valley in Georgia at the eastern end of the Black

Sea. There can be no doubt that scientific nomenclature for pheasants was derived from the geographic names Phasis and Colchis.

The bird was mentioned by many Greek writers, including Aristophanes, Aristotle, and Ptolemy Philadelphus (Pollard, 1977), but Zeuner (1963) did not accord it much importance in the Greek world.

Pheasants were known to the ancient Romans too, but Pliny was among the first to mention them indicating a late arrival, probably after the acquisition of peafowl (Zeuner, 1963). They are included in the Roman writings about poultry, with descriptions of care and housing and the use of eggs and meat.

Zeuner (1963) credits the Romans with distributing pheasants throughout their empire. He described Roman art works from Germany and North Africa which depicted the bird. According to Delacour (1977), the birds of the Romans were from the black-necked group of subspecies. They were well established in France and in England by the tenth century, forming the basis for the populations existing there even now. Zeuner (1963) noted that these birds were never fully domesticated; they were kept exclusively as a game bird for the wealthy.

Many introductions to Europe from Asia were made beginning in the 18th century. Delacour (1977) recorded the introduction of Prince of Wales pheasant (white-winged group) to England in 1902 where it was absorbed through hybridization. The Kirghiz (*P. c. mongolicus*) came to England in 1900. Two of the grey-rumped subspecies, both of them ring-necked, became vastly successful as introduced game birds in Europe; the Chinese ring-necked pheasant (*P. c. torquatus*) reached England in the 18th century and was very common throughout Europe by the 19th century; the Taiwan ring-necked subspecies (*P. c. formosanus*) was brought to Europe in 1907 and it too spread rapidly. Long (1981) has provided a very detailed review of these and other introductions in European countries. The addition of all of these to the black-necked stock of Roman days has resulted in a very heterogeneous European population.

North American introductions have likewise been numerous and have involved many subspecies (Long, 1981). The first to be successful involved birds from China released in the Willamette Valley of Oregon in 1882. Recorded introductions have included at least six subspecies of *P. colchicus* (*bianchii, colchicus, mongolicus, persicus, talischensis, and torquatus*) and have also included the green pheasant *P. versicolor*.

Semidomestic stocks currently raised in confinement for meat production are very recent derivations from those introduced and bred for sport shooting. Their precise origins have not been recorded and it can be assumed that they trace to the same progenitors.

DOMESTIC DUCK

It has been accepted since antiquity that domestic ducks were derived from the wild mallard (*Anas platyrhynchos*). They were domesticated first in the Far East in very early times. A second major domestication occurred in Europe probably during the Middle Ages.

Wild Species

Classification. Mallard ducks have been classified by Howard and Moore (1984), Johnsgard (1978), and Scott (1985) as order Anseriformes, suborder Anseres, family Anatidae, subfamily Anatinae, tribe Anatini which are the dabbling or surface-feeding ducks, genus and species *Anas platyrhynchos* (Linnaeus) 1758. Johnsgard (1978) listed seven subspecies:

A. p. platyrhynchos - common mallard
A. p. conboschas - Greenland mallard
A. p. fulvigula - Florida mallard
A. p. maculosa - mottled mallard
A. p. diazi - Mexican mallard
A. p. wyvilliana - Hawaiian mallard
A. p. laysanensis - Laysan mallard.

The common mallard is probably the sole progenitor of the domestic form. There are two closely related species that are sometimes grouped with *A. platyrhynchos* under the general term 'mallards' (Delacour, 1956). These are *A. rubripes* (American black duck) of eastern North America, and *A. poecilorhyncha* (grey or spot-billed duck) of India, southeast Asia, and Australia. Both overlap in their present ranges with *A. platyrhynchos*, although this may be a recent event. Hybrids among all three species have been reported (Gray, 1958); it is known that hybrids between *A. platrhynchos* and *A. rubripes* are fertile, but reproductive capability of other crosses has not been reported.

Description. Detailed descriptions of the mallard duck have been given by Delacour (1956) and Johnsgard (1975, 1978) and many others.

In the common mallard and in the Greenland subspecies, plumage pattern and color of the green-headed male and drab brown female, both with iridescent blue secondaries, are so familiar that they need no further description. The wild-type color and pattern are retained in some domestic breeds, for instance in Rouen and Grey Call. Common and Greenland mallard males assume an eclipse plumage during the flightless period when they generally resemble females. In other subspecies and in closely related species, male plumage is much like that of females; consult Johnsgard (1975, 1978) for details.

Body weights of common mallards vary with age and season but usually range from 1.0-1.2 kg. Behavior has been studied intensively; it differs little from that found in domestic birds. The birds are monogamous, a trait lost during domestication, and pair bonding appears to terminate with the onset of incubation. Incubation time is 28 days, as it is in domestic ducks. Mallards adapt to a wide range of habitats, but as dabbling ducks they require shallow-water feeding areas. They prefer dry nesting sites in tall vegetation such as among upland grasses and weeds, in dry marshes, or in hay fields. They migrate from northern areas but will spend the winter wherever open water and feed are available.

Distribution. Common mallards have an extremely wide distribution in the Northern Hemisphere (Johnsgard, 1978). In Europe and Asia the breeding range extends from about 35°N to the Arctic Circle and sometimes beyond. They have a similarly extensive range in North America, except for the eastern Arctic, Quebec, the Atlantic Provinces, and New England. Other subspecies of mallard have very limited distribution. Common mallards have been introduced successfully in eastern North America, Bermuda, Australia, and New Zealand (Long, 1981).

Domestication And Early History

The history of domestic ducks is peculiar in that there is almost no early record of them. In the absence of information, it has been generally assumed that first domestication occurred long ago in the Far East, and that a separate European domestication occurred during the Middle Ages.

Early Far Eastern domestication has been assumed because duck husbandry was found to be far-advanced and artificial incubation was an ancient technique (Landauer, 1951) at the time of first European contact. Yeh (1980), cited by Clayton (1984), stated that there have been domesticated ducks in China for at least 3000 years. Watson (1969) indicated the possibility of even earlier domestication in describing the finding of pottery models of ducks and geese at a Lung Shan site in Hupei (about 2500 B.C.) in company with models of sheep, dogs, turtles, and fish.

Zeuner (1963) has speculated that duck domestication may also have occurred in Mesopotamia from Sumerian times (beginning 2800 B.C.) into the Assyrian period (900-600 B.C.). Numerous figurines perhaps representing ducks have been found including 'duck beads', decorated clay vessels, and 'duck weights'; the latter are of varying sizes, the smaller ones carved from hematite and the larger ones from stone, representing a waterfowl in sleeping position with head tucked away. But Zeuner (1963) cautioned that the figurines could represent either ducks or geese and that all diagnostic details are lacking.

There is no indication that ducks were kept as domestic birds in ancient Egypt (Zeuner, 1963). According to Harper (1972), translations of Egyptian papyri confirm that geese, chickens, and pigeons were being raised from the fifth century B.C. to the seventh century A.D., but ducks were seldom mentioned and any reference probably was to wild fowl.

According to the early writers the Greeks and Romans kept ducks, but indications are that the birds were wild and had to be confined within netting enclosures (Harper, 1972). Cato about 160 B.C. mentioned fattening of hens, geese, and squabs but did not mention ducks. Varro was the first to mention them about 37 B.C., referring to the need for covered enclosures to keep ducks in and predators out. Cicero, Columella, and Pliny the Elder all recommended gathering eggs of wild ducks and hatching them under hens. Zeuner (1963) has suggested that domestic ducks may not have been totally unknown to the Greeks and Romans based on his interpretation of the works of Aristophanes. Chicken and goose bones were found in large numbers during archaeological study of the Imperial Roman town of Gorsium (Bökönyi, 1984), but it is very significant that no bones were positively identified as belonging to domestic ducks. There were 20 specimens from mallards and unidentified *Anas* species but no indication that these were other than wild birds. However, Bökönyi (1984) cited three other studies of Imperial Roman sites where domestic ducks were identified with full certainty, and a fourth study where identification was very probable. The most likely conclusion is that domestication may have been in a very early phase during Roman times in which birds had not yet lost wildness and flying ability. These domestication efforts seem not to have been fully successful since nothing in the recent historical record indicates Greek or Roman origin of present-day domestic ducks.

There is fragmentary evidence of duck domestication by Germanic tribes. Zeuner (1963) has described archaeological finds from a Bronze Age site in Bavaria, a mosaic from Cologne showing a drake among known domestic animals, and bones decorated with ducks or duck heads from Hallstatt-La Tène graves in Germany. None of these offers positive proof of domestication. Harper (1972) has found a few clues in writings from the barbarian age, including Germanic law codes which deal mostly with poaching. He interprets a passage from law of the Salian Franks written in A.D. 798 as making definite reference to domesticated ducks, as does an estate document issued by Charlemagne. By A.D. 810 domestic ducks were listed among the holdings of peasants, although they were greatly outnumbered by geese and chickens. Harper (1972) is careful not to claim first domestication of ducks in Europe, but he does indicate that domestication there was likely independent rather than being borrowed from elsewhere. It probably occurred repeatedly; Darwin (1896) and many others have commented on the ease with which common mallards can be molded into domestic birds. The contribution and importance of these relatively recent domestications to modern domestic duck stocks has not been assessed.

MUSCOVY DUCK

The muscovy duck was domesticated in pre-Columbian times from the wild muscovy *Cairina moschata*. It is probable that many independent domestications occurred, but there is no certain record of such.

Wild Species

Classification. Muscovy ducks were classified by Howard and Moore (1984), Johnsgard (1978), and Scott (1985) as order Anseriformes, suborder Anseres, family Anatidae, subfamily Anatinae, tribe Cairinini, genus and species *Cairina moschata* (Linnaeus) 1758. There are no recognized subspecies.

Description. Detailed descriptions of the wild bird have been given by Delacour (1959), Johnsgard (1975, 1978), Leopold (1959), and Phillips (1922). Wild and domestic forms are much alike, except that the wild birds are smaller and less bulky, and they have uniformly dark plumage. Body weights of wild males are 2.0-4.0 kg and of females 1.1-1.5 kg. Plumage of both sexes is solid brownish black, and in mature individuals there is a green or purple sheen to the feathers. White patches develop on the wings with advancing age, involving under wing coverts, axillaries, and upper wing coverts. Facial caruncles are much less developed in wild muscovies than in domestics and they are not as conspicuously red. Behavior has not had much study in wild birds, but it appears to be the same as in domestics; they are aggressive, promiscuous, and not very social, and they roost at night. Reproductive activity of wild birds has been studied only north of the Equator, where they appear to respond to increasing daylength. Incubation time of wild muscovies is believed to be 35 days, as it is in domestics. The preferred habitat consists of marshes, lagoons, and rivers at relatively low altitudes especially where associated with forests. Wild birds are not migratory, but during dry seasons they may move to coastal swamps or lagoons.

Distribution. Muscovy ducks are native to Mexico, Central America, and most of South America (Delacour, 1959; Johnsgard, 1975, 1978; Leopold, 1959; Long, 1981; Phillips, 1922). Their Mexican and Central American range includes coastal plains southward from central Sinaloa in the west and central Nuevo Leon in the east. They avoid highlands and do not occupy the interior of the Yucatan peninsula. In South America they extend to the coast of Peru in the west and to Santa Fe, Argentina in the east.

They have been introduced many times outside of their normal range (Long, 1981), particularly in American states bordering the Gulf of Mexico. Most introduced populations represent feral domestic stocks. An introduction in New Zealand did not succeed.

Domestication And Early History

It is generally accepted that muscovies were domesticated in the Americas during pre-Columbian times, but there is little information to indicate where and how many centuries before the Spanish Conquest it had occurred. According to Delacour (1964), the Spanish Conquistadores found domesticated muscovy ducks, including color variants, on the northern coast of Columbia and in Peru. Phillips (1922), citing De Armas (1893), identified the Colombian location as Cartagena, the capital of the State of Bolivar, where according to Oviedo domesticated muscovies were being kept by the Indians in 1514. The domestic bird was also abundant in Peru at that time, and from there the Spaniards exported it under the name *pato perulero* to Central America, Mexico, and Europe. Gilmore (1950), cited by Clayton (1984), favored a central Andean region of high culture, probably Peru, as the centre of domestication, indicating that representations of muscovy ducks on pre-Columbian pottery are common there. In Landa's *Relación de las cosas de Yucatán*, written in 1566, it is said that the natives raised a certain kind of large white duck which was believed to have come from Peru (Leopold, 1959). Based on these reports, most authors have assumed that domesticated muscovies did not extend into Mexico until they were brought in from elsewhere by the Spaniards. However, Whitley's (1973) interpretation of other writings could indicate that domesticated muscovies were indeed present in pre-Columbian Mexico where they served important religious and cultural functions in the Aztec civilization. At least one of these reports clearly refers to muscovy ducks, but the wording strongly suggests wild birds whose natural range includes both coasts of southern Mexico. Whether wild or domestic, Whitley's (1973) study reveals that muscovy feathers were valued for making decorative garments. He has also suggested that the red duck-billed mask of the Aztec god-hero Quetzalcoatl was derived from the muscovy duck, but his evidence for this derivation from domestic stock is weak.

Early diffusion of muscovy ducks from the New World has been reviewed by Phillips (1922), Clayton (1984) and others. A special problem encountered is that of very rapid spread of both the birds and an awareness of them following their first recorded discovery in Colombia and Peru in 1514. Jeffreys (1956), cited by Clayton (1984) and by Reed (1977), proposed that muscovy ducks reached Africa in late pre-Columbian times on trans-Atlantic ships of Arab traders, but that theory is not fully accepted by historians. Whether it was pre- or post-Columbian, first entry to Europe was apparently from Africa. Aldrovandi in Italy accurately described and illustrated the bird in 1603, stating that it came from Cairo, and Belon in France also described it well in 1555, referring to it as '*La grosse Cane de la Guinée*' (Clayton, 1984). According to Brown (1929), Caius mentioned it as the Turkish duck in 1670, and Willughby who died in 1672 referred to it as a wild Brazilian duck. Muscovy ducks had reached Taiwan by 1693 where they became known as foreign ducks (Clayton, 1984).

Muscovy ducks have been known by a great variety of names. Brown (1929) listed some of them — Brazilian, Peruvian, Guinea, musk, Muscovite, Turkish, Barbary. He believed that the word muscovy is a corruption of musk duck, so named because of the peculiar odor emitted by old birds. That odor was frequently mentioned by early writers, but it is not known to moderns. The name may have been derived instead from the Muisca Indians of central Colombia (Sauer, 1969). Ligon found them as 'muscovia ducks' in Barbados about 1650 (Clayton, 1984). Moubray's (1854) interpretation is very plausible. The great 16th century merchant companies were responsible for European introduction of many new products from the New World and the Orient. To promote both company and product, they attached company name as prefix, as in 'Turkey coffee' introduced by the

Turkey Company. One of the major merchants during Queen Elizabeth's reign was the Muscovite Company. It may have imported the duck. There has never been any indication that Moscow or anything Russian was involved. Clayton (1984) has provided a further discussion of names and naming, including those used in other languages.

Further historical information on diffusion of muscovy ducks is sadly lacking. They have too often been grouped with domestic ducks (*Anas platyrhynchos*), thereby losing their identity as a separate species. Even now they are usually overlooked in agricultural surveys and census data.

GOOSE

Most authors agree that geese have undergone multiple domestications. There seems to be no doubt that the greylag goose (*Anser anser*) of Europe and Asia is ancestral to the domestic breeds of western origin. The swan goose (*Anser cygnoides*) of Asia is the likely ancestor of eastern breeds but evidence is less compelling. There appear to be no major barriers to interbreeding of the two kinds.

Wild Species

Classification. Geese have been classified by Howard and Moore (1984), Johnsgard (1978), and Scott (1985) as order Anseriformes, suborder Anseres, family Anatidae, subfamily Anserinae, tribe Anserini which comprises swans and true geese. The swan goose is *Anser cygnoides* (Linnaeus) 1758; there are no recognized subspecies. The greylag goose is *Anser anser* (Linnaeus) 1758; two subspecies are known, western (*A. a. anser*) and eastern (*A. a. rubrirostris*).

True geese comprise 14 species, divided into 'grey geese' (*Anser* spp.) and 'black geese' (*Branta* spp.) (Delacour, 1954; Johnsgard, 1978; Scott, 1985). Hybridization within a genus can be generally successful, but hybrids are not always fully fertile *inter se* or in backcrosses to either parent; hybrids between *Anser* and *Branta* can also occur but the progeny are usually sterile (Gray, 1958). Reproductive isolation within genera and species is maintained primarily by behavioral differences and social bonding among flock mates (Scott, 1985). It is evident that cytological differences may also be important. Shoffner's unpublished work (cited by Crawford, 1984) indicated that *Anser* and *Branta* differ mainly in configuration of the fifth chromosome, and within *Anser* spp. there are differences in second and fifth chromosomes.

Description. Detailed descriptions of the wild species ancestral to domestic geese have been given by Delacour (1954) and Johnsgard (1978).

The swan goose (*A. cygnoides*) is the most primitive of the grey geese, but sufficiently like them to warrant inclusion within *Anser*. Its body is long and angular in comparison with greylag geese. It is characterized by a very long black bill, and there is only the suggestion of a knob at its dorsal base, a feature which is strongly developed in African and Chinese breeds. Plumage color and pattern are identical to those in brown varieties of Chinese and African breeds. Body weights are about the same as in greylag geese, males averaging 3.5 kg and females 2.8-3.5 kg. Preferred habitat is in association with rivers and lakes. They are exclusively vegetarian. Behavior is essentially the same as that of other grey geese. Sexual maturity begins during the second winter under both wild and captive conditions. Pair bonding is strong and is retained throughout egg laying, incubating, and rearing of the young. Clutch size is 5-8 eggs, weighing 145 g each. Incubation time is 28 days.

The greylag goose (*A. anser*) is almost chunky and rotund compared with the swan goose. Plumage color and pattern are nearly identical to that of the Toulouse breed. Body weights are 2.8-4.1 kg in males and 2.5-3.8 kg in females; the two subspecies do not differ appreciably in size. They are vegetarian. Preferred habitat is open wetlands and grassy uplands; they feed extensively in cultivated fields whenever there is an opportunity. Behavior has been thoroughly studied, and it has had wide dissemination particularly through the popular writings of Konrad Lorenz. The behavioral repertoire is very similar to that of domestic geese. Pair bonding is strong and of long duration, beginning during the second winter. Clutch size is variable but averages 5-6 eggs which weigh 160 g. Incubation time is 28 days.

Distribution. Breeding range of the swan goose (*A. cygnoides*) extends from southcentral Siberia eastward to Kamchatka and south to central Asia and northern Mongolia; its winter range includes China and Japan (Johnsgard, 1978). There is no record of introductions to areas outside its normal range. Current status of the species is unknown, but it is believed to have severely declined in numbers.

The greylag (*A. anser*) has a much broader breeding range through northern Europe and Asia (Johnsgard, 1978). The western subspecies (*A. a. anser*) breeds in Iceland, Scotland, and from Scandinavia south to Austria and east to the Caucasus. It winters in the Mediterranean area including North Africa. The eastern subspecies (*A. a. rubrirostris*) breeds in a broad band across central Asia from the Urals and the Caspian Sea to Manchuria. In the eastern part of its breeding range it is sympatric with swan geese. It winters in Asia Minor, India, and northern Indochina where it overlaps with wintering swan geese. Because of extinction of local populations, many reintroductions have been made, particularly in Great Britain where they have been generally successful (Long, 1981). Introduced stocks have persisted in Belgium, Holland, and Sweden. Early attempts to establish greylag geese in New Zealand met with failure.

Domestication And Early History

Ancestral species. Based on biological resemblance, there is no reason to doubt that greylag geese were ancestral to the European domestic bird. Wild and domestic forms differ in only very minor ways, they can interbreed freely, and the hybrids are fully fertile. Poultry writings of the late 19th and early 20th centuries frequently include anecdotes concerning introgression from wild birds.

Delacour (1954) stated without elaboration that the western subspecies *A. a. anser* was the ancestral form, and that the eastern *A. a. rubrirostris* was never domesticated. However, historical accounts of goose breeding in Russia (Petrov, 1962; Brown, 1929) describe the practice of hatching greylag goslings from wild-gathered eggs or catching wild goslings to be raised in captivity. At least some of this practice was inside the natural range of the eastern subspecies. It would be surprising if domestication did not follow. Indeed the Russian breeds described by Brown (1929), especially those bred for fighting, were quite different from contemporary breeds of western Europe. Petrov (1962) rejected the view that Chinese geese were derived from the swan goose, believing instead that they came from greylags, and hence from the eastern subspecies. Further investigation is needed to resolve whether *A. a. rubrirostris* contributed to domestic birds.

As early as 1819 it was believed that the swan goose (*A. cygnoides*) was also represented in the domestic bird. Main (1819) thought so and many subsequent authors agreed with him. But some did not, for instance Brown (1929), Tegetmeier (1867), and Petrov (1962). Darwin (1896) avoided the debate entirely. It remained for Delacour (1954, 1964) to be dogmatic in declaring dual origin of domestic geese, and subsequent authors have accepted his opinion.

The main evidence for descent from *A. cygnoides* is identical plumage pattern and color in the wild form and in the domestic African and Brown Chinese. Recent cytological studies have provided strong additional evidence for relatedness. In Shoffner's unpublished work (cited by Crawford, 1984) there was tentative evidence that the Emden (*A. anser*) differs from Chinese and swan goose (*A. cygnoides*) in morphology of the fifth chromosome. Silversides et al. (1988) have confirmed this in a cross of African (*A. cygnoides*) with Pilgrim (*A. anser*), showing that the difference appears to involve an inversion in the fifth chromosome (fourth largest autosome). If an inversion is involved, impaired reproduction in hybrids might be expected. In Gray's (1958) check-list of bird hybrids there is indication that F_1 progeny of *A. anser* x *A. cygnoides* have normal fertility, but hatchability in backcrosses is very low. Further investigation is warranted.

History of domestication. Very early domestication of geese seems to have occurred in the Far East. Zeuner (1963) commented accordingly but could offer no evidence. However, Watson (1969) described pottery models of ducks and geese from a Lung Shan site in Hupei (about 2500 B.C.). They were associated with similar models of sheep, dogs, turtles, and fish providing strong evidence but not full proof of domestication. The Hupei site is within the modern breeding range of swan geese. Delacour (1964) mentioned pictures over 2000 years old depicting obviously domestic heavy birds. Further prehistoric record is sadly lacking.

Zeuner's (1963) review of geese in archaeology and classical literature of the western world is the most comprehensive to be found. Only a few bits of information can be added to it. He believed that Germanic tribes had domesticated the bird very early, perhaps even before the time of the great Mediterranean civilizations. Zeuner's (1963) evidence for Germanic domestication is scanty; it is based on interpretation of a few archaeological relics from the Bronze Age, and on linguistics.

There is no doubt that geese underwent domestication in Egypt too. There is abundant pictorial and written evidence, none of it indicating introduction from elsewhere. Riddell's (1943) speculative account, overlooked by most writers, introduces new interpretive ideas. Wild geese were an important game bird in the Nile delta, they probably were at some time taken into captivity, and then eventually they became domesticated. The Nile delta is within the present winter range of greylag geese but it is not within the breeding range. Presuming that these ranges have not changed in historic times, the process of domestication in Egypt must have been started with wintering adult birds. Geese are well documented from the Old Kingdom (2686-1991 B.C.), including both greylag and white-fronted species, but there is little evidence to indicate that either species was domesticated at that time (Zeuner, 1963), although Boessneck (1960, cited by Bökönyi, 1984) believed that they were. However greylags were fully domesticated by the time of the New Kingdom (1552-1151 B.C.); a mural from Thebes dated about 1400 B.C. illustrates a flock of mixed colors being herded. The Egyptian stocks persisted for many more centuries; Harper (1972) made reference to translations of papyri dating from the fifth century B.C. to the seventh century A.D. which described the keeping of geese, chickens, and pigeons.

The greylag goose was kept as a domestic bird by the ancient Greeks (Zeuner, 1963). They are not mentioned in the Iliad, but they are described in the Odyssey. The Greeks fattened them on soaked grain, eggs were depicted on coins, and the bird was regarded both as a symbol of plenty and as sacred to Aphrodite. Brentjes (1965, cited by Bökönyi, 1984) had found evidence of domestic geese in Asia Minor in the fourth or fifth millenium B.C. These may relate to the later Greek birds and predate by many centuries the Egyptian domestication.

Geese were an important domestic animal in the Roman civilization which followed that of the Greeks (Zeuner, 1963). The Romans consumed both meat and eggs, and they knew how to produce fatty livers. They used goose fat medicinally, and had developed a goose quill pen by the fourth century A.D. They borrowed from a Belgian tribe the practice of plucking birds twice yearly to obtain down feathers for cushions and upholstery. Geese also played an important role in Roman religion. They were associated with Juno, goddess of marriage, and with Priapus, a fertility deity. Eventually they also became associated with Mars, the god of war, after they saved Rome from an attack by the Gauls in 390 B.C. That story concerns a flock of sacred white geese kept in the Temple of Juno; they detected intruders and sounded the alarm; Dixon's (1848) translation from Livy lib. v. cap. 47 comprises 230 words in two short and two very long sentences.

Importance of geese as a source of meat, fat, and feathers to the Romans should be emphasized. Writings from that time contain many references to husbandry of geese and to their uses. They had their own geese on specialized farms described by Varro and Columella (Bökönyi, 1984), and they brought them in droves across the Alps from Gallia Belgica and Germania. According to Zeuner (1963), those from Germania were white and much preferred. Bökönyi (1984) found 310 goose bones in his study of faunal remains from the old Roman town of Gorsium in Pannonia (Hungary) (A.D. 50-350). The bones were clearly from domestic birds because of their great range in size. A high frequency of goose remains was also found at a Roman site in Austria, and at a site on the lower Rhine where geese outnumbered chickens (Bökönyi, 1984).

Most historical studies of chickens indicate that with decline of the Roman Empire, advanced culture of chickens came to an end. Chickens reverted to a role of indigenous scavengers until the agricultural renaissance of the 19th century. Scholars have not considered the status of geese during that dark period. They certainly persisted and were noticed occasionally, as for instance in law codes and estate records in the eighth and ninth centuries (Harper, 1972) and in occasional writings through to the 18th century (Riddell, 1943). Because of their biological attributes and easy husbandry, domestic geese may have fared better than chickens through the Dark Ages. Further investigation by historians is warranted.

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Chapter 2. POULTRY GENETIC RESOURCES: EVOLUTION, DIVERSITY, AND CONSERVATION

R.D. Crawford

Recent generations of poultry students and poultry specialists generally lack awareness of the vast genetic diversity that has accumulated in domesticated bird species. This diversity has tended to become segregated within the realms of special purpose and special interest groups such as industrial breeders and producers, laboratory scientists, aviculturists, hobbyists, and gene conservationists. Initiates to these groups may not be aware of the genetic diversity held by others.

Many of the chapters in this book describe known variants and variation in relation to species, body systems, and production parameters. The purpose of this particular chapter is to consider poultry genetic resources in a more general way, to consider how they have evolved and accumulated, and to consider their future. Conservation and preservation of resources as insurance against future needs has become a topic of mounting concern. It is pertinent to consider that topic here in relation to past and present genetic resources and in relation to perceived needs for the future.

EVOLUTION AND ACCUMULATION OF GENETIC DIVERSITY

Nearly all poultry species seem to have undergone a similar series of evolutionary stages during their history. Throughout, the major forces which alter gene frequencies (mutation, migration, genetic drift, and selection) have been active. Of these, mutation has been a basic requisite. Selection, both natural and artificial, has been primarily responsible for keeping and accumulating the variation which now exists. Migration and genetic drift were imposed unwittingly by man.

No doubt the first changes occurred during the process of domestication. It is important to recognize man's purposes in domesticating birds and the influence these purposes may have had on evolutionary change. The historical record (Chapter 1) indicates that first use was religious and cultural. This has been especially well documented for chickens, but it is also evident in the record for most other poultry species. The birds gained importance in white and black magic, in religious observance, in folklore and in mythology. A probable direct result was selective retention of color and morphological variants. Soon afterwards the birds achieved importance in cultural practices. Feathers and bones were used as tools and in decorative arts. Bird behavior became a source of entertainment, as in Japanese quail kept for song, and chickens kept for fighting. Only much later did man begin to use his domestic birds as a source of food.

A second evolutionary stage for all poultry species was coincident with and resulted from diffusion outward from the centres of domestication to other countries, other

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continents, other cultures, and other environments. The process of diffusion brought genetic drift and migration into prominence as forces altering gene frequency, and subsequent geographic isolation led to development of distinctive regional types. Diffusion also stimulated natural selection for adaptation to new environments, and a modicum of artificial selection to meet new needs and objectives.

The third stage was typified by the 'hen craze' of the 19th century (Crawford, 1984a; Skinner, 1974). It coincided with the explosive expansion and growth of industry and agriculture in Europe and the Americas. Poultry which until then had lived mostly as domestic scavengers became immensely popular, their monetary value increased greatly, and selective breeding began in earnest. But it is curious that this sudden interest in poultry was almost exclusively cultural. Only very minor attention was given to eggs and meat as food products. Poultry breeding and keeping became a favored hobby of royalty and the upper classes. Huge sums of money were spent in acquiring breeding stock both locally and from abroad, competitive showing was started, and distinctive breeds and varieties proliferated, all with the objective of perfecting feather and form. Most breeds and varieties in existence now were developed in that era.

The fourth stage belongs to the 20th century. It grew out of the cultural 'hen craze' into the vast poultry meat and egg industry of today (Crawford, 1984a; Warren, 1974). Two independent events early in the century, rediscovery of Mendelian principles of heredity and invention of a reliable trapnest for measuring egg production, helped immensely. Throughout, the developing industry has been very quick to adopt new advances in genetics and animal breeding, and new advances in technology. Production poultry were first selected and bred as purebreds. Between 1930 and 1950 crossbreds began to be used. Since then strain crosses, particularly for egg production chickens, have become prevalent. The numbers of breeds, varieties, and strains used in food production have declined to the very few which now dominate the poultry industry. Concurrently the numbers of individual breeders and breeding companies also declined and now only a few multinational corporations supply most of the production-bred birds to the world's poultry industry. It would appear that artificial selection has been the most active evolutionary force during this last stage, yielding poultry with astounding performance in the production of eggs and meat.

Divergence within poultry species as they moved through these evolutionary stages has been variable. Some have changed very little from the wild ancestors. In others, the accumulated variation has become vast.

Chickens. The earliest archaeological evidence for chicken domestication (Chapter 1), both from China and from the Indus valley, is an increase in skeletal size. A seal was found at Mohenjo-Daro depicting fighting cocks, which may be evidence of selective breeding. The written record of Aryan occupation of the Indus valley indicates that by 1000 B.C. chickens had importance in religion and eating of them was forbidden.

Literary and pictorial records from early times in Persia and Mesopotamia, Greece, Egypt, and Italy indicate accumulation of variants in color, morphology, behavior, and body size (Chapter 1). The classical writers of Greece knew of various breeds, and their descriptions indicate the presence of genes for black plumage, rose comb, and beard. The pea comb heterozygote from 1450 B.C. in Egypt is strikingly like modern Asiatic chickens in appearance (see Figure 1.1). Literary record from the Roman Empire indicates further accumulation of diversity. Chicken bones among faunal remains at a Roman military post in Hungary showed that one of the stocks being raised was twice the size of another.

Aldrovandi's chapter on chickens (translated by Lind, 1963) provides a summary of chicken variation that was known by about A.D. 1600 in Europe. It is a remarkable document, both for its encyclopedic text and for its woodcut illustrations. He provided an inventory of types or breeds, both contemporary and known to the ancients. Seven very old kinds were listed: Adrianic, Tanagra, Lydia, Rhodes, Chalcidice, Media, and Alexandria. They varied in size and appearance, and some were known for their fighting ability. He described bantams or dwarfs that were known to Pliny and Aristotle, and that were contemporary among the Italians, British, and Flemish. He knew of Indian chickens that were triple the size of Italian chickens. And he described variants now known as silky, duplex comb, rumpless, and feathered feet. All of these are clearly evident in the woodcut illustrations along with crest, rose comb and frizzle.

Europeans encountered chickens nearly everywhere they went as they explored the 17th and 18th century world (Chapter 1). But detailed descriptions of these stocks are generally lacking. Chickens with black feathers, meat, and bones were known in Africa and in India. Those of Central and South America are claimed to have had Asiatic origin but some of them were certainly introduced from Spain. Although poorly documented, presumably chickens of northern Europe were already much larger than those of the south. The gigantic fighting stocks of southeast Asia were becoming known, but the docile heavy chickens of China remained isolated for another century.

The book by Main (1819) has been strangely overlooked by historians. It is a treatise on French poultry husbandry prepared for British readers. It includes an inventory of chicken breeds or types that were known to Europeans early in the 19th century (Table 2.1). The list is longer than that of Aldrovandi from 1600, indicating both development of local breeds and the discovery of others in faraway places.

Darwin's (1868, 1896) inventory is better known (Table 2.2). He believed that most of the existing breeds had already been imported into England although many sub-breeds were probably still to be found. His list is more concise than the list of Main (1819), but it does not necessarily represent less genetic variation.

These two inventories mark the end of the second evolutionary stage, where genetic

Table 2.1. Breeds or types of chickens known to Main (1819).

Known to the Ancients:

- Adria
- Alexandria
- Bantam
- Woolly or Downy Fowl of Japan
- Chalcidia
- Media
- Tanagra
- Rhode

Contemporary:

- Bahia
- Camboge
- Caux
- Five-clawed and Six-clawed
- Half-India or Java
- Fleche
- Frizzled
- Hamburg
- Tufted
- Isthmus of Darien
- Jago
- Lombardy
- Dwarf
- Negro and Fowl All Black
- Large Footed
- Persian
- Philippines
- Fowl Without Feathers
- Sansevarre

drift and migration were the prominent evolutionary forces, and they also mark the beginning of the third phase, the 'hen craze' era of the 19th century, when most of the breeds and varieties known at present were developed and perfected. It is noteworthy that neither inventory includes the few breeds which now dominate the chicken meat and egg industry of the world.

The 'hen craze' era spanned the late 19th and early 20th centuries. It had a profound effect on assembling and segregating genetic diversity in domestic fowl. There is a voluminous literature from that period, much of it in the form of encyclopedic treatises on all aspects of poultry culture, but especially concerning breeds and breeding. They contain a wealth of historical information that has received little attention from modern scholars. The largest collection of this literature has been assembled in the James E. Rice Memorial Poultry Library at Cornell University. Moubray's (1815) *Treatise on Domestic and Ornamental Poultry* appeared in at least ten editions and was immensely influential; his real name was John Lawrence. Dixon's (1848) *Ornamental and Domestic Poultry: Their History and Management* was also revised and reprinted many times, but it was widely criticized for lacking in factual information; the Rev. Dixon, Rector of Intwood-With-Keswick, was more scholar than poultryman. Tegetmeier's (1867) *Poultry Book* appeared in several editions; his work is of particular interest because of his close association with Charles Darwin. Wright's (1873) *Illustrated Book of Poultry* is probably the best known of them all; it was revised and reissued many times and in many countries. And there were many other authors and books from that time.

All of these authors described the origins and development of the vast array of breeds and varieties that persist to the present. Their descriptions indicate breed formation from local stocks of Britain and Europe, and the tremendous influence of stocks brought from China and the Far East, which evolved into breeds now known as Cochin, Langshan, and Brahma, but which also contributed significantly to development of many other breeds.

There is strong but poorly documented indication that the Oriental stocks also had a major influence on the dual purpose breeds which emerged in the United States (Fox, 1987, personal communication). The whaling industry at the time centred on seaports of the New England coast. Returning vessels sometimes arrived carrying livestock acquired as food supplies in foreign ports, and these remnants were sold on arrival. A cursory inspection of specimens in seaport museums has indicated that the chickens were from the Orient but remarkably like present-day American dual purpose chickens.

The breeds which currently dominate the world's poultry industry were all developed during the 'hen craze' era. Chickens from Leghorn (Livorno) in Tuscany reached the United States about 1830 (Skinner, 1974) and they were popular in England by 1876 (Brown, 1929); the Single Comb White Leghorn has replaced all others as a producer of industrial white-shelled eggs. Eggs with brown shells are commercially produced from crosses of

Table 2.2. Chief breeds of chickens according to Darwin (1868).

Game
Malay
Cochin or Shangai
Dorking
Spanish
Hamburgh
Crested or Polish
Bantam
Rumpless
Creeper or Jumper
Frizzled or Caffre
Silk
Sooty

several dual purpose breeds developed after 1850; the major contributors are Plymouth Rock, New Hampshire, Rhode Island Red, and Australorp. Modern broilers are based heavily on a cross of Cornish and White Plymouth Rock. The Cornish was developed in England from Asiatic fighting stock, and the White Plymouth Rock was derived as a mutant of the American parent breed.

The fourth stage in evolution of domestic fowl is in progress now. It is the subject of many chapters in this book, particularly those of Parts III, IV, and V.

Turkeys. Domestication of turkeys is very recent, nearly all of it occurring within the time span of recorded history (Chapter 1). They were in transition from cultural use to food use at the time of the Spanish Conquest. The domestic birds were smaller than wild turkeys, and there were color variants among them. They remained relatively unchanged for several centuries. Darwin (1896) could list only a few kinds distinguished mostly by plumage color — Norfolk, Suffolk, White, and Copper-colored or Cambridge. Early in the 20th century, Brown (1929) could add only a few more. The 'hen craze' phenomenon had little effect on turkey breeds and breeding.

The first dramatic alteration occurred in eastern North America. Domestic turkeys brought from Europe by the early settlers hybridized with the native eastern wild turkey (*Meleagris gallopavo silvestris*). Resultant progeny were much larger than either parent, and they had the bronze plumage of the *silvestris* parent rather than the black appearance of the original Mexican domestic birds. The hybrid was so desirable that introgression from *M. g. silvestris* became a general phenomenon, resulting in a stock which became known as American Bronze. Many were exported. They eventually replaced most of the little Mexican birds in poultry flocks throughout Europe. But except for size and coloring, they differed little from the ancestral forms.

Development of broad-breasted turkeys, which revolutionized the turkey industry, is credited to an English gamekeeper, Jesse Throssel (Davey, 1974; Small, 1974). He had been selecting turkeys for meat quality, hatchability, and early maturity. He immigrated to Canada in 1926, and in 1927 imported a tom and two hens of his own stock. They produced 75 progeny. He was unable to sell them as breeding stock in Canada because they did not conform to accepted color standards. And so he exhibited some in northwestern United States where they created a huge demand and sold briskly. Soon virtually all North American breeding stocks were being selected for broad breasts and heavy muscling, using the English-Canadian stock and using simple mass selection within existing American stocks. All of the modern turkey industry is based on these selections.

Other galliforms. Japanese quail evolved as a domestic form within the past 800 years. There may have been introgression from wild birds into the domestic population since World War II. Their brief history is given in Chapter 1. Their first use was cultural. Only in very recent decades has breeding for egg and meat production become prominent. Recent laboratory breeding has resulted in accumulation of many mutations (Chapter 13).

Guinea fowl have changed hardly at all from the wild ancestor (Chapter 1). Stocks are being selected now in some parts of the world for meat production and selection progress has been rapid.

Ring-necked pheasants present a peculiar case. They were domesticated or semidomesticated very long ago, but only as a game bird for sport hunting. Only now are some being selected for meat production under intensive husbandry.

Waterfowl. The historic record on domestication of ducks and geese is scanty (Chapter

1). There is evidence that all domestic species first served cultural needs, and only later found use as food producers.

The major breeds of domestic ducks seem to have been developed long ago. They were made more uniform during the 'hen craze' era but otherwise they were unchanged. For instance Moubray (1854) described Rouen, Aylesbury, Call, Tufted, and East Indian breeds. Darwin (1896) knew of these and he also knew of the Penguin duck recently imported from southeast Asia that soon became known as the Indian Runner. The Pekin breed, now the predominant breed for industrial meat production, had reached England by 1872 and it arrived in the United States in 1873 (Brown, 1929).

The same situation prevailed with geese. Eastern domestic geese evolved into two distinct types, an active light-weight kind now called Chinese, and a very heavy variety now called African. The two reached Europe from many different locations in Asia and Africa, but despite these varied geographic origins they were remarkably uniform and have remained so to modern times. According to Brown (1929), Chinese geese were known in North America by 1788 and they were well-known in England by 1848. By that time three kinds were recognized, one white, and two brown, presumably the African and two Chinese varieties. The Western breeds of today also have a very long history, although perhaps only as color variants. Darwin (1896) knew of the grey Toulouse, the white Emden, and the curly Sebastopol, all of considerable antiquity. Moubray (1854) had earlier described Emden, Toulouse, pied or saddleback, and Brown and White Chinese.

Muscovy ducks, the most recently domesticated of waterfowl species, have undergone very little change. There are a few color variants and there is variation in body size, but otherwise they still have close resemblance to the wild ancestor.

ACCUMULATED GENETIC VARIATION

There are many ways in which the genetic variation that has accumulated during centuries of domestication can be catalogued. It can be described in terms of breeds, varieties, strains, and lines developed for many different purposes: birds bred for exhibition and recreation, birds bred for food production, those bred for laboratory research, and those which are 'unimproved' and indigenous. It can also be described at the level of chromosomes and genes. And it can now be described at the molecular level. Each of these will be considered in turn.

Exhibition breeds and varieties. Nearly all of the recognized breeds and varieties of poultry in western countries can be credited to the breeding and selection work of exhibition breeders. They began their work during the 'hen craze' era and they continue now as hobbyists (see Chapter 41). Some of the breeds and varieties which were synthesized or consolidated then have remained as exhibition stocks. Others have been further selected as egg and meat producers, and they now form the basis for industrial production of eggs and meat. In many western countries, descriptions of ideal standards for recognized breeds and varieties have been published, and they are revised as needed. The best know are *American Standard of Perfection*, *American Bantam Standard*, and *British Poultry Standards*.

A listing of the standard or large chicken breeds recognized by exhibitors in Canada and the United States is given in Table 2.3. Many of the breeds are subdivided into varieties, distinguished by differences in comb type and/or in plumage color and pattern. Nearly all of them can be seen each year at major poultry shows. A tremendous array of bantam or miniature chicken breeds and varieties also exists. Many are tiny models of the standard breeds, but a few exist only in bantam form. They are listed in Table 2.4. Exhibition breeds and varieties of turkeys, ducks, and geese are much fewer in number. They are listed in Tables 2.5, 2.6, and 2.7. Guinea fowl have never been very popular among exhibition breeders, nor have ring-necked pheasants and Japanese quail, although each species has a dedicated following.

Similar lists could be constructed for other countries. Such lists would contain many of the same breeds, although there may be a few unique to particular areas.

Fighting stocks. Through the ages man has found enjoyment in the sport of cock fighting. It is a subculture that is little known to outsiders, but it can be found throughout the world. The sport is illegal in many countries because of uncontrolled gambling and humane considerations, but it flourishes in others. It attracts all levels of society. The birds, usually males, are fought in pairs and the loser is killed in combat. The winner will sire the next generation of fighters.

There is an extensive literature on cock fighting, but it is difficult to access. Most treatises discuss breeds and breeding. The most extensive treatment of these topics is given

Table 2.3. Standard chicken classes and breeds (and varieties) recognized by exhibition poultry breeders (APA, 1983).

AMERICAN
- Buckeye
- Chantecler (2)
- Delaware
- Dominique
- Holland (2)
- Java (2)
- Jersey Giant (2)
- Lamona
- New Hampshire
- Plymouth Rock (7)
- Rhode Island Red (2)
- Rhode Island White
- Wyandotte (9)

ASIATIC
- Brahma (3)
- Cochin (9)
- Langshan (2)

CONTINENTAL
- French
    - Crèvecoeur
    - Faverolle
    - Houdan (2)
    - La Flèche
- North European
    - Campine (2)
    - Hamburg (6)
    - Lakenvelder
- Polish
    - Polish (10)

ENGLISH
- Australorp
- Cornish (4)
- Dorking (3)
- Orpington (4)
- Redcap
- Sussex (3)

MEDITERRANEAN
- Andalusian
- Ancona (2)
- Catalana
- Leghorn (16)
- Minorca (5)
- Sicilian Buttercup
- Spanish

ALL OTHER STANDARD BREEDS
- Game
    - Modern Game (9)
    - Old English Game (13)
- Oriental
    - Cubalaya (3)
    - Malay (5)
    - Phoenix (2)
    - Sumatra
    - Yokohama (2)
- Miscellaneous
    - Araucana (5)
    - Frizzle (2)
    - Naked Neck (4)
    - Sultan

in the excellent monograph by Finsterbusch (1929), also difficult to obtain but apparently available in reprint form in Australia (Plant, 1986, personal communication). Most of the fighting birds in western countries were derived from British stock, which also evolved into the exhibition breeders' Old English Game. But a vast array of other types from many countries is also utilized regionally and internationally. They differ both in morphology and in agonistic behavior. No descriptive inventory except that of Finsterbusch (1929) is available.

Because success in combat requires both agility and strength, it should not be surprising to find that the fighting stocks are well endowed with breast and thigh muscling. Neither should it be surprising to find that nearly all commercial broiler chickens were derived in part from the Cornish breed, which evolved from the Cornish Indian Game, which in turn was derived from fighting cocks of India.

Industrial egg and meat production stocks. FAO (1973) and Crawford (1984a,

Table 2.4. Bantam chicken classes and breeds (and varieties) recognized by exhibition poultry breeders (APA, 1983).

GAME BANTAMS
- Modern Game (12)
- Old English Game (19)

ROSE COMB
CLEAN-LEGGED BANTAMS
- Ancona
- Antwerp Belgian (9)
- Dominique
- Dorking
- Hamburg (6)
- Leghorn (6)
- Minorca (2)
- Redcap
- Rhode Island Red
- Rhode Island White
- Rosecomb (3)
- Sebright (2)
- Wyandotte (10)

ALL OTHER COMBS
CLEAN-LEGGED BANTAMS
- Araucana (5)
- Buckeye
- Chantecler (2)
- Cornish (5)
- Crèvecoeur
- Cubalaya (3)
- Houdan (2)
- La Flèche
- Malay (5)
- Polish (10)
- Shamo (3)
- Sicilian Buttercup
- Sumatra
- Yokohama (2)

SINGLE COMB CLEAN-LEGGED
OTHER THAN GAME BANTAMS
- Ancona
- Andalusian
- Australorp
- Campine (2)
- Catalana
- Delaware
- Dorking (2)
- Frizzle
- Holland (2)
- Japanese (6)
- Java (2)
- Jersey Giant (2)
- Lakenvelder
- Lamona
- Leghorn (10)
- Minorca (3)
- Naked Neck (4)
- New Hampshire
- Orpington (4)
- Phoenix (2)
- Plymouth Rock (7)
- Rhode Island Red
- Spanish
- Sussex (3)

FEATHER-LEGGED BANTAMS
- Booted (6)
- Brahma (3)
- Cochin (13)
- Faverolle (2)
- Frizzle
- Langshan (2)
- Silky (4)
- Sultan

1984b) classified poultry stocks which are kept for egg and meat production in several very broad categories — industrial, middle-level, indigenous, and feral. The categories reflect ownership and utilization, and they also imply certain breeding systems and genetic stocks peculiar to each. The same categories will be used here.

Industrial stocks are those bred by gigantic multinational corporations for use worldwide in mass production of eggs, both white-shelled and brown, and of chicken broilers, turkeys, and broiler ducks. Breeding and selection procedures are detailed in Chapter 40. The genetic resources being utilized are of particular interest in this chapter. Precise details of genetic composition of the industrial stocks are closely guarded corporate secrets, but generalities are public knowledge.

White egg layers. Virtually all are White Leghorns, comprising three-way or four-way crosses of selected grandparent lines. Only a few corporations (primary breeders) share most of the industrial market for the world, and their numbers continue to decline through mergers and acquisitions. It is believed that not all of the grandparent lines are fully unique to each primary breeder; some of the corporations may be utilizing the same grandparent sources. Crossbred parents are placed with multipliers who generate the final commercial pullet for industrial egg production.

Brown egg layers. These are bred in the same way as white egg layers, and by the same primary breeders. The genetic base is broader, utilizing Rhode Island Red, Barred Plymouth Rock, Australorp, and New Hampshire among others. Some primary breeders also incorporate White Leghorn in the commercial product to yield a pale or tinted egg shell.

Chicken broilers. Most of the world's commercial broilers are a four-way cross. The paternal grandparents are synthetic lines derived in part from White Cornish. The maternal grandparents are also synthetic lines; they are based heavily on White Plymouth Rock. Other breeds may be used to generate chicks with colored feathers for particular markets. Only a few primary breeders dominate the market; their numbers are also decreasing.

Turkeys. Even fewer primary breeders supply turkey poults to the industrial market of the world. The poults are usually a three-way or four-way cross. The grandparent stocks are all broad-breasted and nearly always white-feathered.

Waterfowl. Industrializing of duck meat production is not as far advanced. Most of the ducklings for industrial production are strain crosses based almost exclusively on the White Pekin breed. Industrial breeding of geese is just beginning.

Middle-level production stocks. These are the traditional stocks that were the basis for poultry production in developed countries prior to replacement by industrial stocks and industrial production. They have reasonably good productive performance when kept under traditional husbandry conditions. They comprise the dual purpose chicken breeds,

Table 2.5. Turkey varieties recognized by exhibition poultry breeders (APA, 1983).

Beltsville Small White	Narragansett
Black	Royal Palm
Bourbon Red	Slate
Bronze	White Holland

'unimproved' turkeys and ducks, and all of the goose breeds. They are used as purebreds or as breed crosses, especially by small holders and subsistence farmers. They seldom are subjected to intensive genetic selection. Most of them have disappeared from the market in developed countries.

Indigenous stocks. Native or indigenous stocks are those that have persisted in the village economies of long-settled countries for centuries. They live mostly as scavengers with minimal care. Productive performance is very low. They are presumed to have evolved genetic adaptation to local environmental conditions, and hence may have value in developing local stocks with improved performance, as in the case of Fayoumi and Deshi chickens. No comprehensive description and global inventory of indigenous stocks has ever been undertaken, although there have been detailed studies of a few regional populations. Literature pertaining to indigenous stocks, particularly of chickens and ducks, is reviewed in Chapter 42.

Feral stocks. Feral refers to domestic animals which have returned to the wild and away from the care of man. They are considered by gene conservationists to have value because of demonstrated adaptation to the natural environment. Unlike some livestock species, poultry species have only seldom been able to establish permanent feral populations. Darwin (1896) knew of a few feral chicken stocks on oceanic islands, but there has been no recent survey to determine whether any of these persists. The only recently documented population is that of Northwest Island in the Australian Great Barrier Reef (McBride et al., 1969); the birds are of Japanese origin and may have been feral since 1899. Feral guinea fowl are known to persist in Haiti, Cuba, and Dominican Republic, there are feral populations of muscovy ducks in American states bordering the Gulf of Mexico, and Japanese quail introduced to the Hawaiian Islands in 1921 may actually be feral rather than wild (Chapter 1).

Inbred and specialized lines. Poultry scientists have developed many unique genetic stocks for research purposes. They include inbred lines, stocks containing particular blood type alleles, lines carrying mutant genes and chromosome rearrangements, randombred control strains, and others. Somes (1988) listed 217 of these special stocks in chickens, 66 in Japanese quail, and 14 in turkeys. Many of them are also listed in the handbook of inbred and genetically defined strains of laboratory animals prepared by Altman and Katz (1979).

Several chapters in this book discuss in detail the development and utilization of these

Table 2.6. Duck classes and breeds (and varieties) recognized by exhibition poultry breeders (APA, 1983).

HEAVY WEIGHT
- Aylesbury
- Muscovy (4)
- Pekin
- Rouen

LIGHT WEIGHT
- Campbell
- Magpie (2)
- Runner (8)

MEDIUM WEIGHT
- Buff
- Cayuga
- Crested (2)
- Swedish

BANTAM (DUCK)
- Call (4)
- East India
- Mallard

stocks. Inbreeding and inbred lines are described in Chapter 39 and in the monograph by Festing (1979). The use of specialized lines in research on immunogenetics and disease genetics is discussed in Chapters 22 and 33. Randombred control strains and their use in poultry breeding are the subject of Chapter 38.

Poultry geneticists who have been concerned with maintaining and studying mutations have sometimes purposely established them in populations which segregate for mutant and normal alleles. This has been done to ensure uniformity of background genome when measuring mutant effects, and to provide full-sib experimental controls. Examples include muscular dystrophy (*am*), epileptiform seizures (*epi*), naked neck (*Na*), and their normal alleles, and alternate blood types (*Ea-B*). Many of these segregating lines are discussed in subsequent chapters, particularly in Chapter 20.

Gene mutations and cytogenetic changes. Genetic variation in poultry can also be catalogued at the level of chromosomes and genes. All of the chapters in Part II concern these variations.

Very large numbers of mutations have been described in chickens. An alphabetical listing was published by Somes (1980) and it has been revised several times in his series of international registries (1981, 1984, 1988). The 1988 edition listed about 350 mutant loci. It also listed about 100 for Japanese quail and about 50 for turkeys. All of these are described in the chapters which follow, along with lesser numbers of mutations known in other poultry species.

Cytogenetic changes and linkage relationships are the subjects of Chapters 19 and 21. A lot has been learned about domestic fowl, but study of other poultry species lags far behind.

Molecular genetic variation. The very recent development of molecular genetics and genetic engineering technology now permits exceedingly detailed description of genetic variation in DNA and molecular components. These topics are discussed in Chapters 23 and 24. Accumulation of knowledge about variations has been rapid. Table 23.1 presents a list of over 125 genes which have been cloned from chickens. Further major advances in molecular genetics knowledge can be anticipated in the future.

Table 2.7. Goose classes and breeds (and varieties) recognized by exhibition poultry breeders (APA, 1983).

HEAVY WEIGHT
- African
- Emden
- Toulouse (2)

LIGHT WEIGHT
- Canada
- Chinese (2)
- Egyptian
- Tufted Roman

MEDIUM WEIGHT
- American Buff
- Pilgrim
- Saddleback Pomeranian (2)
- Sebastopol

CONSERVATION OF POULTRY GENETIC RESOURCES

"It may soon become one of the implied responsibilities of any organizations or institutions which control the genetic destiny of a whole species to maintain a reserve of variation for further improvements and for unforeseen shifts in the environment or in demand. Indeed, it may be said that each generation has an obligation to see that genetic variation, like soil fertility, is not handed on to its successors in an exhausted state" (Lerner and Donald, 1966).

The conserving of animal genetic resources, particularly those not currently favored by breeders, has become a topic of increasing concern to agricultural scientists, to administrators and planners, and to the general public. The concern relates mostly to genetic insurance against future needs, both for food production and for biomedical application. Most of the discussion, planning, and activity in animal germplasm conservation has pertained to the major livestock species. Relatively little attention has been paid to poultry. But because industrializing of poultry production has proceeded so rapidly, with consequent discarding of genetic stocks having inferior productive performance, the need for conservation is probably greater for poultry species than for any other domestic animals. In the following pages, the need for conservation of poultry genetic resources will be assessed, the conservation activity thus far will be summarized, and planning for the future will be considered.

Arguments for and against conservation. Most of the literature on resources conservation includes discussion of justification. The main arguments were expressed by Maijala (1970) and they have been utilized by many authors since then.

Past losses may have been detrimental. It has been estimated that in Canada there were about 800 breeders of middle-level poultry before the advent of industrial stocks bred by multinational corporations (Hunton, 1983, personal communication). In 1980 there were only 13 (Crawford, 1984b), and most of those are gone now. So are the stocks that they bred. The same has happened in other developed countries. The indigenous stocks in developing countries are also disappearing following invasion of improved stocks from elsewhere (Chapter 42).

Resources are needed for future changes in the environment. A general concept in modern animal breeding is that of fitting animal genotypes to a chosen environment, rather than modifying the environment to suit the animal. Interest has been expressed in cage housing of turkey breeding stocks as a production economy and convenience, but it might be questioned whether genetic variation to permit adaptation of birds to that environment exists within the very narrow genetic base of industrial turkeys.

Resources are needed for future changes in products. Two new products, the chicken sandwich and chicken cubes, have appeared recently in the catering trade of western countries. They have had a dramatic effect in increasing consumption of chicken. These products will also have had an influence on the kind of broiler chicken being bred to meet that particular market. Similarly, there is increasing demand, by those opposed to factory farming, for meat from birds raised under more natural conditions. Some breeders are purposely utilizing the naked neck (*Na*) gene to help meet this demand.

Man cannot create new useful genetic variation. He never could, and he still cannot. Genetic engineering technology may bring some changes in the future.

Avoid monotypic populations and genetic vulnerability. Animal breeders have paid almost no attention to the threat that monotypic populations might be developing and bringing with them vulnerability to genetic (and other) disaster (Crawford, 1984a, 1984b).

The problem is one of long standing and major concern to plant breeders (NAC, 1972). The corn leaf blight epidemic of 1970 in the United States is a widely known example. A single source of cytoplasm had been used in developing most of the corn hybrids being grown there. A new fungal pathogen adapted to that cytoplasm suddenly appeared, and the resulting epidemic reduced the corn yield by 15 percent nation-wide and by 50 percent in some southern states. Major steps have been taken to correct the situation since it was found that most American field crops had become similarly monotypic and vulnerable to genetic disaster.

Monotypy and vulnerability are becoming evident in the industrial poultry sector. Only a few world-class breeders monopolize the breeding of industrial layers and chicken broilers, and it is speculated that some of their grandparent lines may have the same origins. Fewer primary breeders share most of the commercial market for turkeys; one of them is acknowledged to hold over half of the available world market, and it has a near monopoly in some areas. It can be argued that there is strong risk of genetic disaster. It can also be argued that countries such as Canada, which no longer have a national poultry breeding industry, are thus also made vulnerable to economic and political disaster.

Monotypy and vulnerability can also occur at the single gene level. The present commercial chicken industry makes very extensive use of a few major genes such as dominant white (*I*), recessive white (*c*), silver and gold (S, s^+), sex-linked recessive dwarfism (*dw*), slow and fast feathering (K, k^+), and yellow skin (*w*). Linkage associations and pleiotropic effects of these genes are reasonably well known (Chapters 20 and 21), but there can be surprising new findings. The recent discovery of close linkage between slow feathering and an endogenous virus (*ev-21*) associated with susceptibility to lymphoid leukosis (Bacon et al., 1988) has resulted in a move by some breeders away from feather sexing as a means of separating males from females at hatching.

The cost of conservation is very high. Undeniably, it is. The only satisfactory means of conserving poultry genetic resources at present is as living flocks, which are expensive to feed and maintain and to reproduce. Long-term preservation of eggs and embryos is not yet feasible. Sperm cells can be frozen and stored with variable success, but only the homogametic genome is preserved, and according to Buckland (1979, personal communication) very heavy selection pressure is imposed on donors for ability of sperm cells to withstand freeze-storage and thawing. These aspects are discussed further in Chapter 4. The storing of DNA is worth considering, even though it is not yet feasible to utilize stored DNA in living animals.

The probability of future utilization is low. In the first years of conservation concern and activity, this argument and the argument of cost were the strongest deterrents. There of course can be no assurance that a conserved stock will ever contribute anything worthwhile in the future. Neither will a stock which has gone to extinction. The very long and laborious procedure of crossing and repeated backcrossing, to insert a desired trait into a preferred strain, and then to retrieve the original performance level, is not to be undertaken lightly. A great hope for the future is that genetic engineering technology and expertise will permit excising of specific DNA fragments, and insertion of them into preferred strains, without having to resort to traditional breeding procedures.

Literature, conferences, and task forces. A large body of literature pertaining to conservation of animal genetic resources has accumulated. There have been two general forums. There have been symposia, seminars, and special conferences devoted entirely or in part to conservation concerns. The major ones are listed in Table 2.8. Only the 1964 conference in Bologna pertained exclusively to poultry. Proceedings have been published from each conference, those for the European Association of Animal Production (EAAP) meetings appearing in volume 11 of *Livestock Production Science*.

The second major forum has been that of the Food and Agriculture Organization of the United Nations. It has been a major leader in promoting and coordinating interest and action. In the first phase of its work on animal resources conservation, a series of consultations was held to assess problems, to attempt inventories, to carry out pilot projects, and to gather expertise. One of the study sessions, in 1973 at Nouzilly, concerned poultry (FAO, 1973). All of this work was summarized and assessed at a technical consultation held in 1980 in Rome (FAO, 1981). The second phase of its work has consisted of the matters listed in Table 2.9, most of which have been accomplished. Nearly all of it has concerned cattle and sheep, but poultry was included in protocol and procedures for data banks on animal genetic resources information; descriptors to characterize stocks of seven poultry species were prepared for use along with similar descriptors for the major livestock species (FAO, 1986).

As a result of these activities, many have hoped that an international board on animal genetic resources would be established, modelled after the existing International Board on Plant Genetic Resources. It serves as a global coordinating agency, working with regional and national boards in actively conserving plant genetic materials. Thus far, a world animal agency has not been founded. However, several task forces have been actively pursuing that kind of objective. The U.S. Council for Agricultural Science and Technology issued a major report (CAST, 1984) calling for a national program to coordinate the management of animal germplasm resources, and encouraging the development of international programs. Similarly, the Office of Technology Assessment prepared a report for the United States Congress on needs and technologies for maintaining biological diversity of plant, animal, and microbial resources (OTA, 1987). As a culmination of all the assessing, planning, and discussing by groups, organizations, and individuals throughout the world, the U.S. National Academy of Sciences established in 1986 the Committee on Managing Global Genetic Resources. Its purpose is to develop a global strategy for management and conservation of resources, both plant and animal. A series of reports is anticipated in the very near future (Ballachey, 1988, personal communication).

Inventories of poultry genetic resources. The general procedure recommended by the Nouzilly conference (FAO, 1973) on poultry conservation was to prepare an inventory

Table 2.8. Major conferences concerning animal genetic resources conservation.

1959	Chicago	AAAS Germ Plasm Resources Symposium
1964	Bologna	2nd European Poultry Conference
1974	Madrid	1st World Congress on Genetics Applied to Livestock Production
1979	Japan	1st SABRAO Workshop
1982	Madrid	2nd World Congress on Genetics Applied to Livestock Production
1982	Debrecen	International Conference on Gene Reserves
1982	Leningrad	EAAP Annual Meeting
1983	Madrid	EAAP Annual Meeting
1986	Lincoln	3rd World Congress on Genetics Applied to Livestock Production
1989	Coventry	Rare Breeds Survival Trust International Conference

of stocks existing in a given area, collect those which appear to be endangered, evaluate them, and then actively conserve those deemed to be most worthy of maintaining. The procedure is ideal, but seldom very practical. However, at least some conservation action has occurred.

Inventories have been compiled in Australia, the United States, Canada, and perhaps in other countries. A special report was prepared in Australia (SCA, 1979) to assess the need for conservation of poultry stocks; it recommended both cataloguing and conservation. As a result, inventories were prepared and published by the governments of five Australian states (Stanhope, 1984, personal communication).

The excellent series of registries prepared in the United States by Somes (1972, 1975, 1978, 1981, 1984, 1988) has had very wide circulation and use. Listings are volunteered and hence they are far from being a complete inventory, but they still represent a major assessment of genetic resources. The first issue in 1972 included American listings only. The second in 1975 included a Canadian inventory. Subsequent issues have included contributions from many countries.

A specialized inventory entitled *Catalogue of Poultry Stocks Held at Research and Teaching Institutions in Canada* was first issued by the author in 1967, and it was revised each year to the 14th edition in 1981. At that time it was melded with a similar catalogue on laboratory animals prepared by Canadian Council on Animal Care. The combined catalogue was revised and reissued at two-year intervals under the title *Research Animals in Canada* (CCAC, 1985). It is no longer being printed, but the data bank is maintained and revised regularly, and information can be obtained through direct enquiry.

A very detailed national assessment of Canadian poultry breeding stocks at all levels of classification (industrial, middle-level, exhibition, indigenous) was made by Crawford (1984b). Most poultry producers were utilizing industrial stocks, patronizing nearly all of the world-class primary breeders. Middle-level poultry had nearly vanished. He located only 11 pure lines of middle-level chickens where a few decades ago there had been 800, one stock of bronze turkeys with natural mating ability, and a few remnants of middle-level waterfowl stocks. Exhibition poultry was flourishing among hobbyists, but the usual source of stock was a few dealers and supply houses. The original Chantecler, the only recognized breed developed in Canada, was extinct. Except for gene reserves in public agency collections, and those held by an industrial primary breeder, the entire country was found to be impoverished of poultry breeding resources.

Similar inventories may have been made in other countries. If they have not, they should be encouraged. They are especially needed in developing countries where indigenous stocks are rapidly being replaced with industrial poultry from elsewhere. The limited

Table 2.9. Objectives in Phase II of the FAO/UNEP project on conservation of animal genetic resources. (Adapted from FAO, 1984).

Publication of 'Animal Genetic Resources Information' journal.
Appointment of an expert panel of scientists/advisors.
Training courses in conservation methodology.
Survey of indigenous livestock breeds of USSR.
Pilot data banks in Africa, Asia, and Latin America.
Pilot conservation schemes for indigenous breeds.

information available on these indigenous stocks is discussed in Chapter 42.

Collection, evaluation, and conservation. Despite repeated urgings and recommendations from the literature to conserve rare stocks, relatively little real conserving is being done. The practical problems are many, among which are costs and uncertainty of future utilization. Many of the breeders and research workers who have listed stocks in the various registries are motivated by conservation concerns, and they try to keep their stocks to that end, but they must also make current use of them to justify their retention. Only in a few instances can resource collections be regarded primarily as conserves.

There are said to be large resource collections in Russia, Czechoslovakia, and Rumania, but detailed information is lacking. Some rare breeds and specialized lines are being maintained in Europe (Mérat and Hann, 1979). According to Stanhope (1984, personal communication), rare stocks of some poultry species are being conserved at several locations in Australia.

Unfortunately the security and continuance of such programs seems never to be secure. A tragic example is that of the very large collection of former production lines that was held at Parafield Poultry Research Centre in Australia. To ease financial difficulties, a conservation trust was formed, with commitment and contribution from both government and industry (Polkinghorne, 1984, personal communication). The trust operated for only a few years and then it closed, partly because of funding problems; the resource collection has been dispersed (Pell, 1988, personal communication). There are two large resource collections in Canada, but neither has assured security. The collection of Japanese quail at University of British Columbia is probably the world's largest. It operates perforce as a current resource, generating operating funds from sales of produce and breeding stock, and from grant funds for active research projects (Cheng, 1988, personal communication). The collection of middle-level poultry strains and mutations kept by the author at University of Saskatchewan has very little external funding. It survives because of its use as a teaching resource and because it is the medium for frequent publications and publicity.

Except for brief notations in registries and inventories, stocks in conservation collections have usually had little descriptive or metric evaluation. The exception may be that of indigenous stocks, as described in Chapter 42. Availability of standardized descriptors, which were developed for use in animal genetic resource data banks (FAO, 1986), should encourage more rigorous evaluation.

Nearly all of the existing conservation stocks, whether as gene pools, pure breeds, or as specialized lines, are being maintained as living diploids. Techniques for long-term storage of gametes and zygotes are not yet suitable or available for conservation work. Frequent reproduction, large populations, and minimal selection, both natural and artificial, are needed to avoid inbreeding and genetic drift. Where possible, using the procedures for reproduction of randombred control strains (Chapter 38) is to be recommended. Hopefully in the near future techniques for storage and utilization of DNA will become available.

Responsibility for conservation. There has been controversy over who should assume responsibility for conservation of poultry genetic resources as insurance against future needs. Crawford (1984b) considered that three groups — primary breeders, hobbyists, and public agencies — should take part, but that they should not be held equally responsible.

At least some of the primary breeders are holding genetic reserves for future changes in their products. But their business projections are for ten years in advance, not for several decades, and not for a century. They may be held responsible for extinction of genetic resources that were less productive than their own stocks. But for business reasons they cannot afford to expend funds on conservation schemes that have no certain future rewards.

Perhaps poultry hobbyists should be encouraged to undertake the task. Some have argued that the exhibition stocks are being very safely held by the hobbyist fraternity now, and that no further action is needed. However, Crawford (1984b) found in Canada, and Skinner (1974) found in the United States, that most hobbyists are really not breeders; their stocks are frequently renewed from a few dealers and supply houses, implying that the genetic base for exhibition stocks is very narrow. Crawford (1984b) also concluded that the objectives of gene conservationists and exhibition breeders are quite different. Both want to ensure retention of rare stocks. But the conservationist is concerned about genetic purity of line and genetic purity of descent, while the exhibition breeder is more concerned with phenotype. He is not restrained in how conservation is brought about; indeed the ethics of exhibition poultry breeding encourage the use of adventurous crossbreeding to achieve desired perfection of bird specimens. Accordingly, Crawford (1984b) argued that conservation of rare stocks should not be left exclusively to exhibition poultry hobbyists. Carefoot in Chapter 41 has provided strong rebuttal.

A new hobbyist movement, concerned specifically with conservation of animal genetic resources, has emerged in recent years. Membership organizations are now functioning in several countries. Interests include maintaining flocks of relic poultry breeds, particularly those formerly bred for food production. Objectives are similar to those of gene conservationists. It is too early to assess their contribution but it has considerable potential importance.

Perhaps ultimately the major responsibility should rest with public agencies: federal and regional governments, research institutions, and universities. Only they can provide the continuity and assured funding for a long-term conservation scheme. The role of hobbyists and primary breeders should not be discounted, but they cannot be expected to fully assume responsibility for the conservation of genetic resources.

The paradox is that thus far public agencies have done little except discuss the problems of conserving poultry genetic resources. The real activity has been among the hobbyists. It has also been a private activity of senior personnel, both active and retired, of primary breeder corporations.

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Chapter 3. REPRODUCTIVE BIOLOGY IN RELATION TO BREEDING AND GENETICS

H.P. Van Krey

In this chapter, selected topics in avian reproduction are addressed. Topics chosen are considered to be those of most interest and concern to geneticists and poultry breeders. Focus is on the germ cell and the differentiation and development of male and female reproductive systems. The latter are followed through to sexual maturity. In addition, spermatogenesis, oogenesis, fertilization, egg formation, and sperm-oviduct interrelationships are discussed. No attempt has been made to be exhaustive with respect to any particular topic, the intent of the author being to place the subject into perspective and to meet informational needs of those who are not specialists in reproductive biology.

EARLY EMBRYONIC DEVELOPMENT

Primordial germ cells. The earliest morphologically recognizable embryonic cells antecedent to mature sex cells in domestic fowl are the primordial germ cells (*cellulae germinales primordiales*) (PGCs). Ginsburg and Eyal-Giladi (1987) suggested that PGCs may already be determined in preovipositai (Stage X) embryos (staging by Eyal-Giladi and Kochav, 1976), and that further differentiation of PGCs may occur independently of the embryo forming process. Pardanaud et al. (1987) confirmed the preovipositai determination of PGCs, utilizing monoclonal antibodies to identify PGCs in the unincubated blastoderm of *Coturnix* embryos.

Migration of primordial germ cells. As the blood islands and primitive blood vessels are forming in mesodermal tissue, the PGCs that had migrated from their epiblastic (*epiblastus*) site of origin to the region of the germinal crescent, enter the developing blood vessels. Once blood flow is initiated (approximately 33 h incubation) the PGCs circulate temporarily (Stages 11-16; staging by Hamburger and Hamilton, 1951) within the embryonic and extraembryonic circulatory systems. Subsequently, the PGCs escape from blood vessels (Stages 15-18) located in the splanchnic mesoderm (*mesoderma splanchicum*) immediately caudal to the omphalomesenteric artery (Ukeshima et al., 1987) and migrate to the nearby gonadal anlagen via amoeboid movement (Kuwana et al., 1987). Migration of PGCs is influenced by a chemotactic substance originating in the gonadal anlagen, probably the superficial epithelium of the gonadal ridge (Kuwana et al., 1986).

Gonadal anlagen. In the chick embryo, the gonadal anlagen are located initially in the dorsal splanchnic mesoderm on both sides of the dorsal mesentery, where they appear as local thickenings of the coelomic epithelium (Stage 16). Spatial relationships between the site at which PGCs initially settle, the gonadal anlagen and the future gonadal ridges (*crista gonadalis*), have been established (Ukeshima et al., 1987). Accordingly, upon leaving the blood vessels in the splanchnopleuric area, the PGCs colonize the nearby thickened coelomic splanchnic epithelium, and the newly colonized PGCs and thickened splanchnic epithelium subsequently migrate as a unit (Stages 17-18) to the future gonadal ridge. Thus, Ukeshima et al. (1987) suggested that the thickened splanchnic epithelium of the coelom which originally incorporated the PGCs, and only later became the superficial epithelium of the gonadal ridge, should be regarded as the real gonadal (germinal) epithelium (*epithelium superficiale*).[1]

Indifferent stage of embryonic development. After settling in the gonadal ridge epithelium, the PGCs begin to differentiate forming the gonadal epithelium. Prior to 96 h of incubation, the distribution of PGCs is relatively uniform between the gonadal epithelium on the right and left sides of the embryo, but by approximately 96 h of incubation the distribution of PGCs becomes decidedly asymmetrical. More PGCs colonize the left (70 percent) than the right (30 percent) gonad, and an active right to left migration of PGCs occurs in both sexes of *Gallus* but not in *Anas*. The underlying cause of the unequal distribution of PGCs is uncertain. Nevertheless, at this stage of embryonic development, the PGCs remain bipotential, still being capable of differentiating into either male or female sex cells (Maraud et al., 1986).

While differentiation of the gonadal epithelium is occurring, the underlying *mesonephric* mesenchyme (*mesenchyma*) is initiating formation of the rete tissue. *Rete cords* and medullary tissue form in the indifferent gonad, presumably by condensation of the mesonephric mesenchyme, for a deficiency of mesonephric tissue can cause subnormal gonadal development (Rodemer et al., 1986).

Shortly after the rete cords have begun development, an initial proliferation of medullary sex cords occurs. The medullary cords, also known as primary sex cords, first appear as bud-like extensions of the gonadal epithelium (Stages 27-28, 5.5-6 days of incubation), and then separate from that epithelium (Stage 29, 6.5 days of incubation). Sex cords contain a combination of germ cells and sustentacular cells (*cellulae sustentacularis*, also known as Sertoli cells). After approximately seven days of incubation (Stages 30-31), a layer of developing connective tissue, the primary *tunica albuginea* isolates the sex cords and associated stroma from the gonadal epithelium. Formation of the primary tunica albuginea marks the end of the development of the indifferent gonad. Subsequent development is variable, dependent upon the genetic sex of the embryo.

***H-Y* antigen, gonadal differentiation and sex determination.** Genotypic sex is determined by the chromosomal constitution of the zygote. Whereas in *Mammalia* the male is the heterogametic sex, in *Aves* the female is the heterogametic sex, expressed symbolically as *ZW*, and the male is the homogametic sex, expressed symbolically as *ZZ*. In mammals, differentiation of the testes is generally attributed to the expression of the plasma membrane protein that is serologically detectable as *H-Y* antigen, and in birds, differentiation of the ovary is attributed to expression of the serologically cross-reactive *H-W* antigen (Wachtel, 1983). Because the *H-Y* and *H-W* antigens are cross-reactive serologically, the two antigens are perceived as being representative parts of functionally disparate molecules. Evidence exists indicating there has been phylogenetic conservation of the gonadal *H-Y*/*H-W* receptor, which would be expected since genes of fundamental importance tend to be conserved *in toto* and seldom undergo evolutionary alteration (Ohno, 1978).

Based on its broad phylogenetic conservatism, the H-Y (H-W) antigen has been proposed to be the inducer of primary sex, causing the heterogametic gonad to become a testis in male-heterogametic species and an ovary in female-heterogametic species (Wachtel, 1983). Although evidence implicating the *H-Y* antigen in sexual differentiation is extensive, inconsistencies such as *H-Y* negative human males and *H-Y* positive human females exist. More recent data suggest that *H-Y* antigen is not the testis-determining factor (Roberts, 1988), and a possible revision of current concepts regarding gonadal organogenesis may occur (Page et al., 1987).

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[1] According to *Nomina Anatomica* (*Acta Embryologia*), the traditional name, germinal epithelium, is misleading and inappropriate.

SEXUAL DIFFERENTIATION IN MALES

In males, following formation of the primary tunica albuginea, the gonadal epithelium regresses and ceases to differentiate any additional sex cords. Subsequent gonadal development is directed towards the formation of a definitive testis.

Seminiferous tubules. After the primary tunica albuginea separates the gonadal epithelium from the primary sex cords, the sex cords represent the principal component of the differentiated male gonad. In mammals, differentiating sustentacular (Sertoli) cells initially surround the PGCs and then establish the contour of the developing seminiferous tubules. Additional newly differentiated sustentacular cells interconnect with existing cells elongating the seminiferous tubules and enveloping the PGCs. Although not demonstrated experimentally, it is assumed that a similar developmental scheme occurs in *Aves*.

PGCs remain scattered randomly within a developing seminiferous tubule until day 13 of incubation (Stage 39), at which time they begin to differentiate into *spermatogonia*. By day 20 (Stage 45), the newly formed spermatogonia have become aligned as the spermatogenic epithelium (*epithelium spermatogenicum*) which lines the seminiferous tubules. The spermatogenic epithelium of the immature male exists as a single layer of stem spermatogonia along with some nongerminal sustentacular cells which were incorporated into the primary sex cords during differentiation in the indifferent gonad.

Interstitial cells. Randomly scattered interstitial cells (*cellulae interstitiales*, also known as Leydig cells) increase significantly in number through day 17 (Stage 43) of incubation. Morphological changes also occur in the interstitial cells of both sexes at this time (Stages 30-37 in testes; Stages 30-40 in ovaries), with cells progressing from a stellate (mesenchymal) shape to an oval shape (Romanoff, 1960). The cytolocation of the luteinizing hormone receptor complex also changes during morphogenesis, progressing from being on the surface of the cell membrane of stellate-shaped cells to being located internally in oval-shaped cells (Woods, 1987). The transformed cell types exhibit an enhanced capacity for steroid hormone synthesis.

Rete testis. Rete cords, which first appeared during Stages 23-26 in the indifferent gonad, continue to elongate and anastomose in the differentiated male gonad. The rete cords subsequently develop into the *rete testis* of the differentiated male.

SEXUAL DIFFERENTIATION IN FEMALES

Left ovary. In contrast to genotypic male embryos in which only a single proliferation of PGCs occurs, female embryos undergo a secondary proliferation of the gonadal epithelium. However, the right and left gonad (ovary) differ with respect to the extent of proliferation that occurs, which, in turn, affects subsequent gonadal development.

The initial indication of female sexual differentiation is seen in the left ovary. By the eighth day (Stage 34) of incubation the gonadal epithelium of the left ovary begins to thicken, and the PGCs begin active meiotic divisions forming oogonia (*ovogonium*). The underlying medullary tissue of the left ovary differentiates (Stage 35) into two distinct layers, a relatively dense superficial layer and a deeper inner reticular layer. Clusters of oogonia along with some epithelial cells bud from the gonadal epithelium (Stage 35) and then separate from the latter (ca. Stages 36-37) leading to the formation of the secondary sex cords, also referred to as the cortical sex cords. A connective tissue stroma, the secondary tunica albuginea, begins to form on day 14 (Stage 40), which isolates the cortical sex cords from one another, as well as from the gonadal epithelium and from the medulla of the ovary (Romanoff, 1960). Shortly thereafter (Stage 41) the gonadal epithelium and primary tunica albuginea begin to regress.

At this point in development the left ovary of the domestic fowl is demarcated into a distinct highly vascular medulla (*medulla ovarii*) and a cortex (*cortex ovarii*) which contains the ova (Hodges, 1974). Subsequently, grooves develop which separate the cortical tissue into gyri (King, 1975). The gyri become increasingly infiltrated with medullary tissue as the bird matures, essentially obliterating the clear layered demarcation in the adult ovary that had existed between cortex and medulla (Prochazkova and Komarek, 1970; Hodges, 1974; Gilbert, 1979). Instead, two zones or masses of tissue evolve, one that contains primarily germinal tissue and the other primarily vascular and connective tissue for which the names zona parenchymatosa (*zonae parenchymatosae*) and zona vasculosa (*zonae vasculosae*), respectively, have been proposed (Prochazkova and Komarek, 1970). Hodges (1974) prefers that the term cortex be applied to the outer layers of the ovary along with the loose intermediate stromal tissue, and that the term medulla be applied to the vascular core of the ovary.

Right ovary. Embryonic development of the right ovary is similar to that of the left ovary, except it is less extensive. Medullary development is diminished, and development of the cortical sex cords is irregular, ranging from no development to small clumps of PGC-rich cortical rudiments attached to the gonadal epithelium. Additional proliferation of cortical sex cords may or may not occur, and the tunica albuginea is usually not present in the right ovary (Romanoff, 1960). The right ovary generally regresses to a rudimentary vestige after hatch. Nevertheless, if for some reason the left ovary becomes nonfunctional, the rudimentary right gonad can develop into a functional ovotestis, i.e. testicular medullary tissue with tubules containing spermatozoa and cortical tissue which may contain follicles of ovulatory size. The response of the right gonad is dependent upon the age post-hatch that the left ovary becomes nonfunctional.

AN ALTERNATIVE SCHEME OF SEXUAL DIFFERENTIATION

An alternative viewpoint regarding gonadal differentiation has been proposed (Jost, 1972). As opposed to the concept of primary (medullary) sex cords leading to development of seminiferous tubules in males, and secondary (cortical) sex cords leading to ovarian follicles in females, in the alternative scheme of differentiation the mesenchyme of the indifferent gonad is considered to have a generalized organization. The testes differentiate relatively early, whereby the primordial germ cells become enclosed in seminiferous cords comprised of sustentacular (Sertoli) cells. Interstitial (Leydig) cells also differentiate from mesenchymal cells of the blastema. Testosterone from the newly formed interstitial cells and anti-müllerian hormone from the sustentacular cells then lead to the differentiation of masculine external genitalia and the male genital duct system, respectively.

It was subsequently proposed that gonadal organogenesis is directed by the presence or absence of *H-Y/H-W* antigen (Wachtel, 1983). In mammals, testicular organization is under the control of *H-Y* antigen, and ovarian organization results from the absence of *H-Y* antigen. Conversely, in avian species, ovarian organogenesis is under the control of *H-W* antigen and testicular organization results from the absence of *H-W* antigen. Thus, under the influence of *H-Y* antigen, mesenchymal cells of the future testis aggregate relatively early into seminiferous tubules, containing both primordial germ cells and sustentacular cells (Ohno, 1979; Wachtel, 1983). In genetic females, *H-Y* antigen is absent, and, as a result, the ovaries retain the indifferent generalized organization until relatively late in development. While the primary sex cords of the ovary do ultimately develop, their appearance is delayed, and they become fragmented into clusters, followed by the formation of primordial follicles. Because of the absence of testosterone and anti-müllerian hormone, differentiation of external genitalia and genital ducts is of the female type.

The scheme outlined above regarding gonadal organogenesis was developed based on the utilization of mammalian preparations. Comparable detailed studies of progressive morphological changes in the gonads of avian species await completion. Nevertheless, it should be noted that the ultrastructural and high resolution light microscopy study of Pelliniemi and Lauteala (1981) identified an inconsistency in the alternative scheme of gonadal organogenesis. They found that in embryonic porcine gonads, primary (medullary) sex cords developed in both sexes, and the secondary (cortical) sex cords developed in female embryos, an observation that is not consistent with the *H-Y/H-W* antigen theory of gonadal differentiation. Pelliniemi and Lauteala (1981) concluded that additional factors must be involved in gonadal differentiation. The recent work of Page et al. (1987), showing that a testis determining factor maps to a different part of the Y chromosome than does the *H-Y* antigen, seems to agree with this conclusion. Nevertheless, the exact relationship between *H-Y/H-W* antigen and the testis/ovary determining factor should be both interesting and enlightening.

SEX REVERSAL

Sex reversal in females. Exposure of genetic female embryos to grafts of testicular tissue during the indifferent period can induce male gonadal differentiation in the female embryo. This sex reversal remains permanent after hatching (Maraud et al., 1986). Sex reversal is considered to be in response to decreased ovarian estrogen secretion, a result of anti-müllerian hormone secretion by the testicular graft (Stoll et al., 1985).

Interstitial cell populations are greatly reduced in sex-reversed gonads of genetic females, resembling more the populations found in the testes of genetic males (Maraud and Vergnaud, 1986). Because the interstitial cells are a source of estrogen in the female gonad, and the interstitial cells appear to have a primary influence on testis formation in genetic female embryos, Maraud and Vergnaud (1986) hypothesized that normal testis differentiation is dependent upon a similar endocrine mechanism of intrinsic origin. For a review of the pituitary-testicular and pituitary-ovarian axes with respect to embryonic interstitial cell differentiation, see Woods (1987).

***H-Y/H-W* antigen in sex reversal male gonads.** It has long been known that, contrary to the situation in eutherian mammals, sex steroid hormones can influence initial organogenesis in the gonads of nonmammalian vertebrates, including birds. It was also known that early exposure of male avian embryos to estrogenic compounds causes a transient feminization of the male *ZZ* gonad, but it was not known whether the gonadal feminization is related to *H-Y/H-W* antigen. However, in both *in vivo* and *in vitro* studies, cells from feminized *ZZ* gonads tested $H\text{-}Y^{+}$, indicating that exposure to estrogen induces expression of *H-Y/H-W* antigen (Wachtel, 1983).

DEFINITIVE MALE REPRODUCTIVE SYSTEM

The avian male reproductive system is relatively elemental when compared to that of the mammal. The accessory reproductive organs that are common to the latter are absent in *Aves*, which is reflected by the limited quantity of seminal plasma in avian ejaculates. The avian reproductive system consists of paired *testes*, *epididymides*, and elongate highly convoluted *ducti deferentes* which terminate in the cloaca. Within the cloaca are located the associated erectile tissue and a phallus, the morphology of which varies with the species. This cloacal complex is referred to collectively as the copulatory organ (Hodges, 1974).

Testes. Consistent with their internal location in *Aves*, the encapsulating protective tunica albuginea is extremely thin, and does not possess the radiating septa which divide the testis into lobules as in mammals (Lake, 1957). Also consistent with their internal location, the testicular vascular network in *Aves* does not possess a *plexus pampiniformes*. The latter is a vascular countercurrent heat exchange mechanism associated with the mammalian testis which functions to maintain testis temperature below body temperature. Nevertheless, evidence does exist indicating that the relatively high deep body temperature of *Aves* may be detrimental to spermatogenesis.

The seminiferous tubules of the cock testis form an extensive anastomosing network throughout the testis, and they are not discrete as in the mammalian testis (Lake, 1957). The seminiferous epithelium and the interstitial cells located in the interstices between the seminiferous tubules are the two primary tissues found in the testis of the domestic fowl (Hodges, 1974).

Tubuli recti and rete testis. Just before entering the rete testis, groups of seminiferous tubules lose their germ cell lining and coalesce to form the *tubuli recti* (Figure 3.1). The tubuli recti (straight tubules), which are very short, function to connect the seminiferous tubules with the tubular network of the *rete testis*. The rete network, in turn, consists of a series of thin-walled irregular channels embedded in connective tissue. The majority of the rete testis is extratesticular, with a limited amount of the rete network being located intratesticular in *Gallus* and *Coturnix* (Lake, 1981). The rete testis is located on the dorsomedial surface of the testis joining the latter to the *epididymis*.

Epididymis. The rete network opens into the efferent ductules (*ductuli efferentes*) of the epididymis along its total length in domestic fowl and turkeys. The numerous efferent ductules anastomose extensively and then open into the relatively short connecting ductules (*ductuli conjugentes*) which, in turn, empty into the epididymal duct (*ductus epididymalis*). The efferent ductules are derived from the glomeruli and the proximal and distal convoluted tubules of the embryonic mesonephros. The connecting ductules and epididymal duct are derived from the collecting ducts and excretory duct of the mesonephros respectively. The

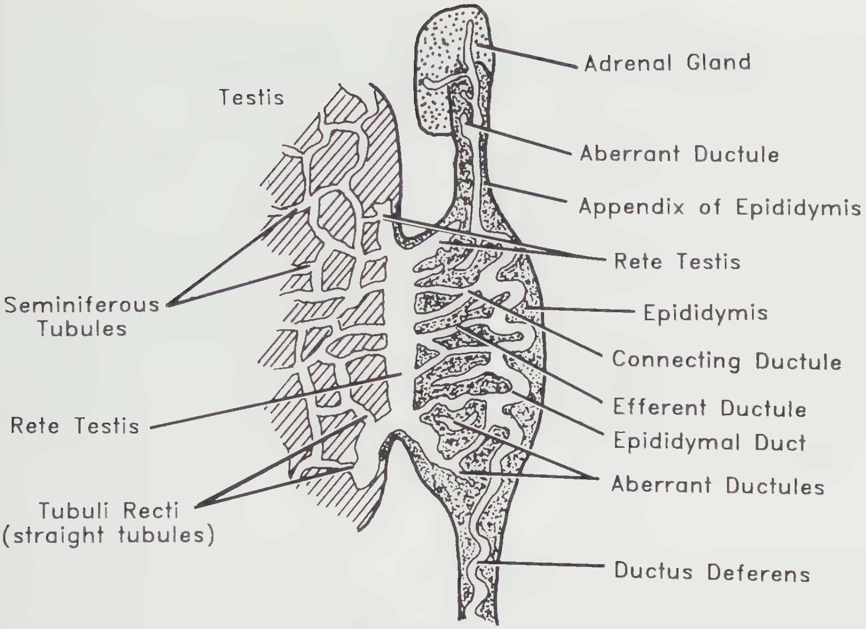

Figure 3.1. Diagram of the testis and epididymis of the domestic fowl. The seminiferous tubules anastomose with each other. The straight tubules are very short and indistinct in histological sections. The rete testis is a network of cavernous channels embedded in the dorsomedial surface of the testis adjoining the epididymis. The numerous coiled and anastomosing efferent ductules arise as wide tubes all along the epididymis and open via the connecting ductules into the epididymal duct. There are numbers of blind-ending aberrant ductules in the epididymis and in the appendix of the epididymis. When the testis is removed the epididymal appendix remains in the body attached to the adrenal gland. (Adapted from King and McLelland, 1984).

epididymal duct in *Aves* is extremely short and the epididymis is not divided into caput, corpus, and caudal regions as in mammals.

Ductus deferens. The epididymal duct joins the ductus deferens together forming the excurrent duct system. The ductus deferens continues on to empty into the *urodeum* of the cloaca via the *papilla ductus deferens*. The ductus deferens is highly convoluted (*ansae ductus deferentis*) throughout its length except for the terminal end (*pars recta ductus deferentis*) which becomes straight just prior to entering the urodeum. The ductus deferens also increases in diameter at this point, expanding to form the so-called receptacle (*receptaculum ductus deferentis*). The ductus deferens is the principal sperm storage organ in male birds. Pressure appropriately applied in the region of the receptacle expells semen during artificial ejaculation.

The excurrent duct system functions in the transportation, maturation, and storage of spermatozoa. It secretes and/or reabsorbs various fluids and materials, including degenerating spermatozoa. Several of these functions, as well as the histology and ultrastructure of the testis and the associated efferent ducts, have been thoroughly reviewed (Lake, 1957, 1981; Hodges, 1974), and will not be covered here. The role of the excurrent ducts on sperm maturation will be discussed later.

Cloacal copulatory complex. A true intromittent phallus is absent from the cloacal complex in *Galliformes*. They possess, instead, a copulatory organ, the *phallus nonprotrudens*, which is located ventromedially in the proctodeum on the ventral lip of the vent (*labium venti ventrale*) (Figure 3.2).

In the domestic fowl the phallus nonprotrudens is comprised of a medial white body (*corpus phallicum medianum*) that is flanked by the paired lateral bodies (*corpus phallicum laterale*) (Lake, 1957; King, 1981a). Located cranially to the lateral phallic bodies are the lymph folds (*plicae lymphaticae*). The phallic bodies and lymph folds become tumescent during copulation. Tumescence is due to the secretion of lymphatic fluid by the paired paracloacal vascular bodies (*corpora vascularia paracloacalia*) (Nishiyama, 1955).

Species-related anatomical differences in the copulatory organ do exist. Also, as opposed to the nonintromittent phallus found in galliforms, the anseriforms possess an intromittent organ, the *phallus protrudens*. For a description of cloacae and the various phalli, the reader is referred to King (1981a, 1981b).

SPERMATOGENESIS

In a newly hatched chick, the basal lamina (*membrana basalis*) of a seminiferous tubule is lined with germinal epithelium. The germinal epithelium is comprised of a mixture of two cell types, spermatogonia derived from PGCs, and sustentacular cells which were incorporated into the primary sex cords along with PGCs during differentiation in the indifferent gonad. Posthatch development of the testes and spermatogenesis has been divided into three general phases by de Reviers and Williams (1984). These phases include the prepubertal phase, which extends to 10-14 weeks of age, the pubertal phase which

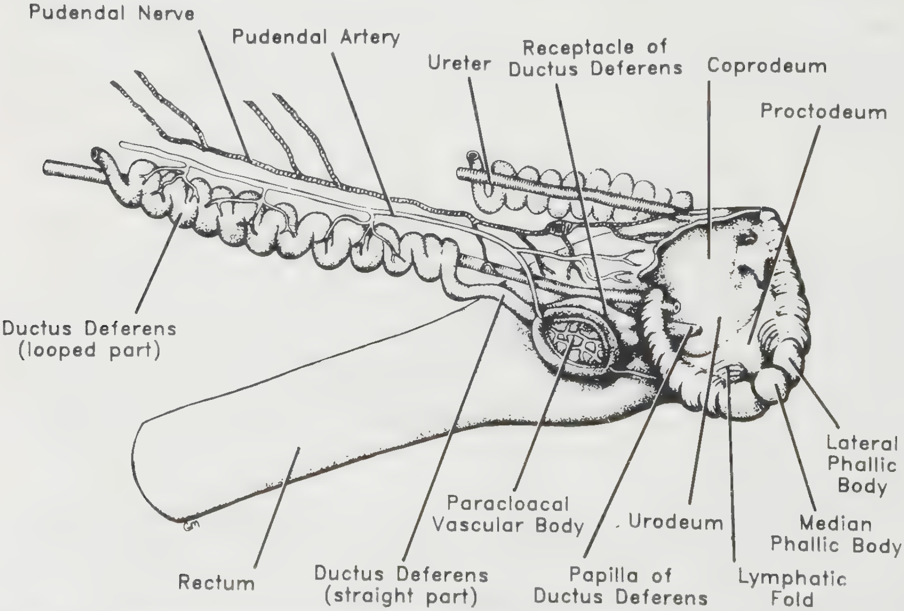
Pudendal Nerve
Pudendal Artery
Ureter
Receptacle of Ductus Deferens
Coprodeum
Proctodeum
Ductus Deferens (looped part)
Lateral Phallic Body
Paracloacal Vascular Body
Urodeum
Median Phallic Body
Rectum
Ductus Deferens (straight part)
Papilla of Ductus Deferens
Lymphatic Fold

Figure 3.2. Lateral view of the cloaca and the terminal part of the ductus deferens of the domestic fowl. (Adapted from Lake, 1981).

extends to 20-24 weeks of age, and sexual maturity.

Testis growth during the prepubertal phase is slow and linearly correlated with both age and body weight. After approximately six weeks of age, increasing numbers of primary spermatocytes (*spermatocyti primarii*) are formed, and the proliferation of undifferentiated sustentacular cells occurs.

The second phase, the pubertal phase, is characterized by a large increase in the rate of testis growth. During this phase, the primary spermatocytes undergo an initial meiotic division leading to the formation of two relatively small secondary spermatocytes (*spermatocyti secondarii*) which, in turn, rapidly undergo a second meiotic division yielding a total of four spermatids (*spermitidia*). Spermatogenesis and the transformation of spermatids to spermatozoa (referred to as spermiogenesis or spermoteleosis) are described in detail by Romanoff (1960), Hodges (1974) and Lake (1981) and will not be addressed here.

The third and final phase of spermatogenesis, sexual maturity, is defined by de Reviers and Williams (1984) as "the termination of testis growth when the number and quality of spermatozoa are at their zenith", as opposed to the initial production of gametes, such as in the hen. This distinction is made because initial semen quality is relatively inferior.

SUSTENTACULAR CELLS AND SPERMATOGENESIS

The nongerminal sustentacular cells have an integral role in the total spermatogenic process. They are apparently involved in the control of spermatogenesis, for daily sperm output and the total number of sustentacular cells per testis are highly correlated (de Reviers and Williams, 1984). Such paracrine control mechanisms, and interactions within and between the seminiferous tubules, the interstitial cells and the testicular vasculature, have been actively investigated in mammals. Similar paracrine testicular control mechanisms have not been extensively investigated in *Aves*.

Sustentacular cells are generally considered to have supportive and nutritive functions within the testes. Sustentacular cells are also involved in steroid hormone biosynthesis, and in phagocytosis of germ cell debris in regressing testes. In mammals, tight junctions between adjacent sustentacular cells are an integral part of a blood-testis barrier. The blood-testis barrier functions to regulate access of some blood constituents to the seminiferous epithelium, maintains concentration differences between the internal fluids and lymph or plasma, and isolates the germ cells immunologically. In domestic fowl, the existence of an effective blood-testis barrier can first be detected in tubules containing early spermatids (Bergmann and Schindelmeiser, 1987).

DAILY SPERM PRODUCTION, DAILY SPERM OUTPUT AND EXTRAGONADAL RESERVES

Sperm production may be defined in two different ways, daily sperm production (DSP) and daily sperm output (DSO). DSP is generally expressed as spermatozoa per gram of testicular tissue. The DSP has been estimated to be 80-120 x 10^6 spermatozoa for adult cockerels (de Reviers and Williams, 1984), 92.5 x 10^6 spermatozoa for Japanese quail (Clulow and Jones, 1982), and 117-135 x 10^6 spermatozoa for turkeys (Cecil et al., 1988). The reported values are quite similar for all three species.

DSO of adult cockerels was found to be 2.28 x 10^9 spermatozoa during peak production (24 weeks of age), but this value diminished to 1.73 x 10^9 spermatozoa by 52 weeks of age (de Reviers and Williams, 1981). DSO for Japanese quail in excess of 8 weeks of age was estimated to be 308 x 10^6 spermatozoa (Clulow and Jones, 1982). For guinea fowl, DSO was found to be 148 x 10^6 spermatozoa at peak production (36 weeks of age), which decreased to 100 x 10^6 spermatozoa per day at 70 weeks of age (Brillard and de Reviers, 1985). The DSO of turkeys (31-42 weeks of age) was reported to be 520 x 10^6 spermatozoa (Cecil et al., 1988), which is less than that of the domestic fowl, but greater than the DSO of guinea fowl and Japanese quail. A geriatric effect on DSO does exist.

Extragonadal reserves. The number of spermatozoa contained in the epididymides and ductuli deferentia is referred to as the extragonadal reserve (EGR). Most of the EGR spermatozoa of the cockerel are stored in the ductuli deferentia, primarily the distal segment of the ductuli (de Reviers, 1972). The epididymides being relatively small, contain only five percent of the EGR.

After the DSP, DSO and EGR have been determined, the EGR may be used to estimate the number of days of maturing spermatozoa being held in reserve by a male. Accordingly, the EGR was found to be .9-19 x 10^9 for domestic fowl (de Reviers, 1972; 1975), 3.2-10.5 x 10^9 for turkeys (Cecil et al., 1988), and 308 x 10^6 for Japanese quail (Clulow and Jones, 1982). These values represent a spermatozoal reserve of 3.5 days duration for chickens, six days for turkeys, but only one day for Japanese quail. Nevertheless, Clulow and Jones (1982) calculated the EGR of the Japanese quail to be adequate for approximately 25 copulations.

Optimum frequency of ejaculation. Quite obviously DSP, DSO, and EGR all affect the optimum frequency of ejaculation when considering a breeding program. It was noted by de Reviers and Williams (1984) that DSO is best exploited by frequent semen collections, and also because semen quality is influenced by frequency of semen collection. Fewer degenerate spermatozoa are found in semen of males ejaculated relatively frequently. In many passerine birds there is a spontaneous continuous release of spermatozoa from the ductuli deferentia into the cloaca (Quay, 1987), which functions to remove aged spermatozoa from the excurrent ducts.

SEMEN

Seminal plasma. The volume of seminal plasma in avian semen is relatively meagre, a reflection of the absence of the accessory sexual glands common to mammals. The limited seminal plasma present in avian semen is derived, in part, from the seminiferous tubules of the testes and from the excurrent ducts. Accessory fluids of cloacal origin are also added to semen during ejaculation. These accessory fluids, also known as transparent fluid, are derived from the lymph folds (*plicae lymphaticae*) (Nishiyama, 1955) and from highly vascularized cloacal mucosa adjacent to and caudal to the ejaculatory papillae.

It has been suggested that transparent fluids may not be a normal constituent of seminal plasma, for these fluids contain ions alien to sperm survival *in vitro*, and contain blood clotting agents which precipitate spermatozoal agglutination *in vitro*. Nevertheless, during natural copulation both spermatozoa and the reducing sugar aldose are transferred from the male to the female (Fujihara and Nishiyama, 1984). Aldose is a normal constituent of transparent fluid, but is not a normal constituent of seminal plasma in the ductus deferens, indicating that it is mixed with the semen during a natural copulation. Seminal plasma used as a semen diluent improved fertility in domestic fowl (Terada et al., 1984; Lake and Ravie, 1987). The latter authors suggested that the composition of a diluent for storage of spermatozoa *in vitro* may be different from a diluent to be utilized as a suspension medium for the transfer of spermatozoa to a female oviduct.

Proctodeal foam gland. In addition to the transparent fluid, a so-called frothy fluid or a foamy secretion may be ejected from the cloaca of the male during mating. A frothy fluid containing protein and glucose is ejected from the cloaca of the male turkey while treading the female prior to mating (Fujihara et al., 1985a; Fujihara et al., 1986a), and a foamy substance is deposited on the female cloaca by males at the time of copulation in Japanese quail (Tamura and Fujii, 1967). The origin of these secretions is the proctodeal gland (*glandula proctodealis dorsalis*), located in the dorsal *proctodeum*. A proctodeal gland also exists in both sexes of the domestic fowl (Komarek, 1970). While the proctodeal gland in domestic fowl is functional, the interval between mounting a hen and copulation is too brief for the male to eject any fluids prior to completing the mating (Fujihara and Koga, 1985). During artificial ejaculation, fluids are ejected from the proctodeal glands of domestic fowl (Fujihara et al., 1985b), but not of turkeys (Bakst and Cecil, 1985; Fujihara et al., 1986a). For a detailed review of the literature and a description of the proctodeal gland see King (1981b).

Ejaculate volume and density. In general, the volume of an avian ejaculate is relatively small and the spermatozoal concentration is relatively great, when compared to that of the mammal. Nevertheless, the volume and concentration of artificially ejaculated semen is influenced markedly by accessory fluids of cloacal origin, i.e. transparent fluid. Density comparisons have been made between ductus deferens and artificially ejaculated semen to obtain estimates of quantity of transparent fluid added to the semen. Accordingly, cloacal accessory fluids dilute semen approximately 50 percent in domestic fowl (Nishiyama, 1955), 20 percent in turkeys (Fujihara et al., 1985), and 50 percent in domestic ducks (Nishiyama et al., 1976). Very little accessory fluid is added to the semen of guinea fowl (Fujihara et al., 1986b). It has been suggested that the extent of semen dilution by accessory fluids may account, in part, for species differences in semen concentration.

Yellow-colored semen. Abnormal yellow-colored semen is occasionally produced by turkeys, and such semen has been associated with poor fertility (Marquez and Ogasawara, 1975). An ultrastructural study of the excurrent duct system of males producing yellow semen revealed abnormal hypertrophied epithelial cells in ductuli efferentes (Hess et al., 1982). The hypertrophied cells contained numerous lipid droplets, the apparent source of the yellow color in the seminal plasma. Phagocytosis of both normal and abnormal spermatozoa was a common occurrence with the ductuli efferentes of males producing yellow semen. It was suggested that stenosis of the ductuli efferentes, the result of epithelial cell hypertrophy, may have precipitated the increased phagocytosis of spermatozoa.

SPERMATOZOA

Numerous studies exist detailing the ultrastructure of avian spermatozoa, and a comparative ultrastructural study (turkey, chicken, guinea fowl) was also recently completed (Thurston and Hess, 1987). As a result of the existing extensive literature, sperm morphology will be characterized here in a general sense only.

Avian spermatozoa show a high degree of morphological diversity. Since this diversity is so great it was suggested that sperm morphology might serve as a basis for speciation (see McFarlane, 1962). Nevertheless, if a broad classification scheme is utilized, avian spermatozoa generally consists of two types: a plain relatively simple type of spermatozoa common to nonpasserine birds, and a more complex type of spermatozoa that is common to passerine species.

Sauropsid-type of spermatozoa. The less complex type of spermatozoa are referred to as the sauropsid-type because of their resemblance to reptilian spermatozoa (Retzius, 1909). Sauropsid-type spermatozoa are elongate flagellated cells measuring approximately 100 µm in length. However, neither the dimensions of the individual segments of a spermatozoon nor the total length are constant either within a species or between species. The configuration of a sauropsid-type of spermatozoon is smooth with the nuclear head (*nucleus spermatozoi*) showing a slight anteroposterior curvature. During motility, the momentum created by the lashing action of the tail is affected by the curvature of the sperm head, resulting in a broad spiraling projectory.

Complex-type of spermatozoa. Sperm cells from passerine species possess a complex helical configuration. They are generally longer (300 µm) than the sauropsid-type of spermatozoa. They possess a relatively large helical acrosome (*acrosoma*), a helical head, and an undulating membrane that extends the length of the spermatozoon (Retzius, 1909; McFarlane, 1962). Motility involves rotation of the entire sperm cell along its longitudinal axis, resulting in forward movement in a corkscrew manner.

DEFINITIVE FEMALE REPRODUCTIVE SYSTEM

Oogenesis. As pointed out by Romanoff (1960) oogenesis (*ovogenesis*) and spermatogenesis are similar in that both eventually lead to the development of haploid cells. Oogenesis differs, however, in that it is characterized by growth and not a loss of protoplasm as in spermatogenesis. Also, the oogenic maturation divisions result in only a single haploid cell, as opposed to four such cells during spermatogenesis.

Oogonia. From approximately day 14 of embryogenesis to shortly before hatch, the female germ cells derived from PGCs undergo a series of rapid mitotic divisions (Romanoff, 1960) yielding many thousands of daughter cells, the so-called oogonia of the definitive ovary. All such oogonia apparently develop prior to hatch, with no additional cells forming posthatch.

Primary Oocytes. At time of hatch, the oogonia have already completed several stages of the meiotic prophase of the first reduction division, and are now defined as primary oocytes (*ovocytes primarius*). Nevertheless, the exact stage of development of primary oocytes at hatch is uncertain. For a complete review of nuclear events in both oogonia and primary oocytes the reader is referred to Romanoff (1960) and Gilbert (1979).

The primary oocyte stage of development has also been referred to as the growth phase and is characterized by yolk accumulation (*vitellogenesis*) in preparation for subsequent ovulation. However, only a very small percentage of oocytes ever reach ovulatory size. Although the fate of any given oocyte cannot be determined, it has been suggested that preferential selection for subsequent growth and development may be occurring (Gilbert, 1971, 1979; Gilbert and Wells, 1984), and that ovarian innervation may be involved in the selection process. The latter authors found that follicular hierarchy may include oocytes considerably smaller than previously thought, extending down to oocytes as small as one mm in diameter. Selective oocyte stimulation by trophic hormones or growth factors based on location relative to large ovulatory follicles may also be involved in the preferential selection process (Armstrong, 1987). For a review of ovarian follicular hierarchy the reader is referred to Gilbert (1971) and Gilbert and Wells (1984).

Yolk (*vitellus*) constituents are synthesized primarily in the liver under the influence of estrogen, transported to the ovary via the circulatory system, where the yolk material escapes the terminal blood capillaries of a follicle and is incorporated into the developing oocyte by receptor mediated endocytosis (Griffin et al., 1984). Vitellogenesis in *Aves* has arbitrarily been separated into various growth phases. In *Gallus*, vitellogenesis is generally recognized as occurring in three distinct phases (Marza and Marza, 1935), an initial period of slow-growth which can be months, or even years in duration, an intermediate-growth phase of approximately 60 days duration, and a rapid-growth phase which is characterized by extensive yolk deposition and the first meiotic reduction division.

During the period of slow-growth, yolk deposition is limited and consists mainly of neutral lipids. The numerous cytoplasmic changes that occur during this stage have been summarized by Romanoff (1960). Nuclear changes that occur at this time are of minimal direct significance to gametogenesis.

Yolk deposition during the next phase, the intermediate phase, is primarily protein along with limited amounts of lipid. Definitive yolk spheres appear initially in oocytes approximately 2.5 mm in diameter (Griffin et al., 1984). Follicular growth progresses to approximately four mm in diameter during the intermediate phase.

The last phase, the rapid-growth phase is of a relatively short duration, the exact time being dependent upon the species. Published values are quite variable, listing the rapid-growth phase to range from 5-14 days, 11-15 days and 5-7 days in *Gallus*, *Meleagris*, and *Coturnix* respectively. However, in a comprehensive study that involved both normal and dwarf White Plymouth Rock hens, Zelenka et al. (1986) found the rapid growth phase to be remarkably constant at 7.5 ± 0.1 (mean ± standard error) days. It would seem that the effects of ambient temperature, which can affect the central:peripheral blood flow ratio, should be taken into consideration when determining the duration of the rapid-growth phase of oogenesis.

Secondary oocytes. The first meiotic division of a primary oocyte is completed only several h prior to the ovulation of that oocyte. However, nuclear changes are noticeable in a primary oocyte approximately 24 h before the first meiotic division occurs (Romanoff, 1960). In *Gallus* the germinal vesicle begins to disintegrate and the nuclear membrane shows evidence of wrinkling. In *Columba* a clear 'polar ring' develops around the germinal vesicle.

After the first polar body (*polocytes primarius*) is extruded, it is visible immediately beneath the vitelline layer (*lamina perivitellina*) as a minute oval globule containing condensed chromatin and limited karyoplasm (Romanoff, 1960). Very shortly after extrusion of the first polar body, and thereby formation of the secondary oocyte (*ovocytes secundarius*), the second meiotic spindle beings to form. However, the second meiotic division progresses only to metaphase and remains at that stage until after ovulation. Completion of the second meiotic division is dependent upon a spermatozoon penetrating the oocyte cytolemma (*cytolemma oocyti*). The second meiotic division generally does not occur in the absence of fertilization.

PARTHENOGENESIS

Parthenogenesis occurs in various invertebrate and vertebrate species of animals. Artificial stimuli, both physical and chemical, can initiate embryonic development indicating that the impetus for development is not necessarily associated with spermatozoa, and the ovum itself may possess such a potential (Romanoff, 1960). While early investigators generally agreed that changes which resembled parthenogenetic development occurred in the blastodisc of some infertile eggs, they disagreed regarding the basis for, and the significance of such development (reviewed by Romanoff, 1960). Nevertheless, parthenogenetic development is now known to occur in both chickens and turkeys.

Most parthenotes fail to hatch, presumably because of abnormal cell divisions which can yield haploid, tetraploid and heteroploid cells (Darcey and Buss, 1968), and because of the frequent occurrence of moribund and dead blastomeric cells (Olsen and Marsden, 1954). Nevertheless, those parthenotes which do hatch are always diploid males (Poole and Olsen, 1957), and experimental evidence suggests that parthenogenetic development starts in haploid oocytes, with the diploid chromosome number subsequently being restored (Darcey et al., 1971; Olsen and Buss, 1972). Because parthenotes are always males, the recombination of a haploid secondary oocyte and a haploid first polar body cannot be occurring, for such a combination should yield some female parthenotes (Olsen and Buss, 1972). Either a suppression of the extrusion of the second polar body, or the re-entry of the second polar body into the newly developed ootid, was suggested by Olsen and Buss (1972) as being a logical explanation for restoration of the diploid state.

As indicated, extraneous stimuli can, on occasion, initiate parthenogenetic development. Olsen (1956) reported vaccination for fowl pox increased the incidence of parthenogenetic development in both chicken and turkey eggs. Also, an outbreak of the disease visceral lymphomatosis (leucosis) increased the frequency of parthenogenetic development in chicken eggs (Olsen, 1966).

THE RIGHT OVIDUCT

In most species of birds, only the left oviduct (*oviductus sinister*) is retained as a functional organ, and the right oviduct (*oviductus dexter*) regresses into a nonfunctional vestige which remains attached to the cloaca. The vestige frequently fills with fluid which is of plasma origin and, on occasion, the vestige becomes quite large occupying much of the abdominal cavity. McBride (1962) investigated the anomaly of dual adult oviducts, and found that inheritance of right oviduct development involved but a single gene. Hens homozygous for the dominant gene (*Ro*) had fully developed right oviducts, whereas heterozygotes showed relatively large cystic right oviducts, and those homozygous for the recessive ro^+ gene usually possessed a small noncystic oviduct remnant. Despite single gene control, the incidence of dual functional oviducts is rare in *Gallus*, as are vestiges of right oviducts in species of wild birds.

THE LEFT OVIDUCT

The left oviduct of the hen is generally recognized as consisting of five functional regions. Progressing craniocaudally, the following regions have been identified: *infundibulum, magnum, isthmus, uterus* and *vagina* (Figure 3.3). Solomon (1983) recognized a sixth distinct region, preferring to separate the uterus (shell gland) into a tubular shell gland and a shell gland pouch.

The wall of the oviduct possesses the same basic structure throughout, being comprised of the following layers: surface epithelium, lamina propria, inner connective tissue, circular muscle, outer connective tissue, longitudinal muscle, and serosa (Figure 3.3). The surface epithelium throughout the oviduct consists of a combination of ciliated cells and unicellular glandular cells. The ratio of those cells is variable, depending upon the region of the oviduct. In addition to the unicellular glands, two types of multicellular glands also exist, tubular glands, and glandular grooves. The tubular glands are found throughout the oviduct, with the exception of the anterior infundibulum (the site of fertilization) and the caudal vagina. Glandular grooves are restricted to the infundibulum (Hodges, 1974).

Located at the junction of the infundibulum and magnum and the uterus and vagina are tubular structures, the so-called sperm nests (*fossae glandulares infundibuli*) and sperm-host glands (*fossulae spermaticae*), respectively. The tubules in both of these areas function to store spermatozoa, a function which will be addressed later.

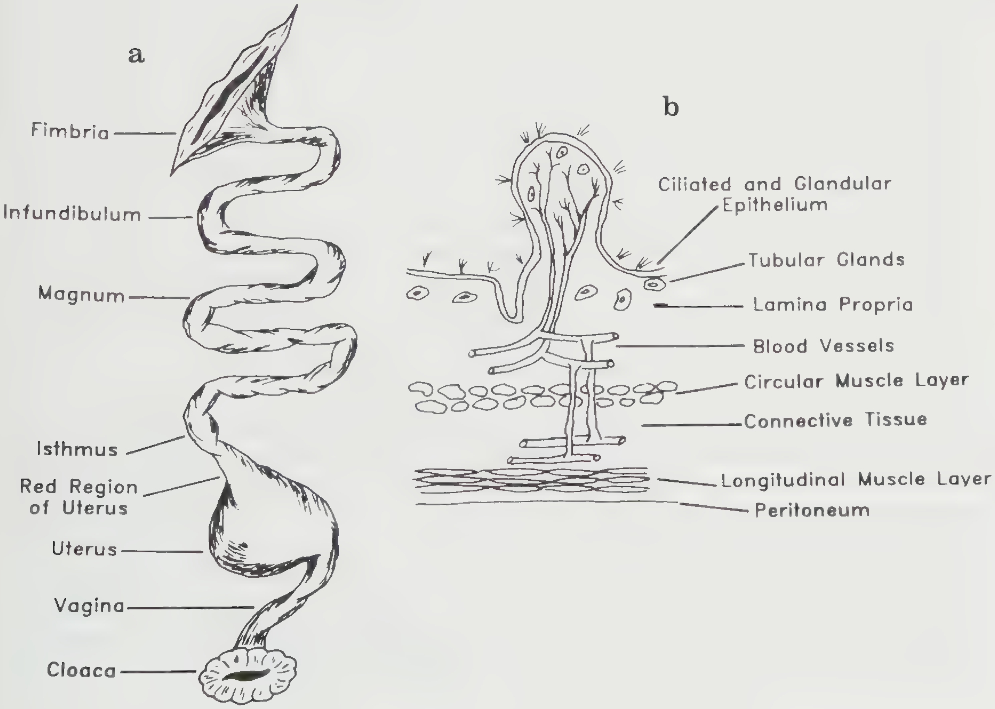

Figure 3.3. (a) Diagram of the oviduct of the domestic fowl. (b) Diagram of the oviduct wall depicting its basic structure. The structure will vary from region to region. (Part b from Gilbert, 1979).

Infundibulum. The infundibulum consists of two distinct regions, a relatively thin-walled fimbriated funnel region (*fimbriae infundibularis*) which functions to engulf a released ovum, and a thick-walled tubular infundibulum (*tubus infundibularis*), which contains tubular glands (*glandulae tubi infundibularis*). Tertiary layers of the total yolk membrane are deposited in the infundibulum, as is albumen in *Gallus*. However, fertilization occurs in the anterior gland-free infundibulum prior to albumen deposition. The ovum spends approximately 15-30 min in the infundibulum.

Magnum. The magnum is the site of albumen secretion and is the largest segment of the oviduct. Mechanical stimulation by the passing egg apparently precipitates albumen secretion. Different secretory cell types have been identified, but attempts to ascribe the secretion of a specific protein to a particular type of cell have been only partially successful (reviewed by Gilbert, 1979; Solomon, 1983). An ovum remains in the magnum approximately 3 h.

Isthmus. A translucent aglandular (1-3 mm) line of demarcation (*pars transluens isthmi*) exists between the magnum and isthmus. The isthmus itself has long been recognized as consisting of two regions based on its gross physical appearance. Draper et al. (1972) divided the isthmus into proximal and distal regions based on secretory cell type. The proximal isthmus contained tubular glands (*glandulae isthmi*) with relatively heavy accumulations of electron-dense granules, while the glands of the distal isthmus contained few such granules. The distal isthmus also assumed a reddish coloration during the periods of vascular engorgement associated with calcium transfer, and was, thus, termed the red isthmus by Draper et al. (1972). Based on histological evidence, it is now accepted that the so-called red region is truly uterine and not isthmal tissue. It has been proposed that the red region be referred to as the tubular shell gland to more clearly indicate its involvement in the shell calcification process (Solomon, 1983). An ovum requires approximately 1.5 h to traverse the isthmus.

The two egg shell membranes (*membrana testae interna* and *externa*), composed of proteinaceous fibres, are formed by the tubular glands in the proximal isthmus. Secretions pass from cells of the tubular glands into the lumena of these glands, where they coalesce and become fibrous in nature before passing into the lumen of the isthmus. The fibres possess an electron-dense inner core and a surrounding less dense mantle.

The so-called mammilary cores of the egg shell matrix have been identified as the epitactic centres of calcite crystal formation (see Parsons, 1982). However, the origin of the mammilary cores is uncertain. Both the isthmus and the red region of the uterus have been proposed as the site of origin. Irrespective of point of origin, it is known that core uniformity, core density, and the morphology of mammilary cores all influence egg shell quality (Parsons, 1982). The remainder of the organic shell matrix is deposited while the egg is in the uterus.

The initiation of egg shell mineralization apparently also occurs in the distal isthmus. However, the limited mineral deposits found on shell membranes of eggs taken from the isthmus are in a form other than the calcium carbonate which is common to egg shells. Thus, the uterus is considered to be the region of mineral crystal growth and not the site of initial crystal deposition.

Uterus. As indicated, the uterus consists of two segments, a short tubular cranial segment (*pars cranialis uteri*) and a pouch-like caudal segment (*pars major uteri*). Despite suggestions and tendencies to utilize the functionally descriptive term shell gland, *Nomina*

Anatomica Avium (Baumel, 1979) prefers the term uterus, noting that it has been in use in avian anatomy for more than 150 years.

Prior to calcification of the developing egg, water and salts are added (plumping) to the albumen, a process requiring approximately six h. Subsequently, the calcite shell, or testa, (95 percent calcium carbonate, five percent organic matter) is deposited during the next 15 h in the uterine pouch. Total time spent by an egg in the uterus is 20-22 h.

Shell pigmentation also occurs in the uterus. The shell pigment is a polycrystalline complex (Solomon, 1983). Pigments for red-brown (*oöporphyrin*) and blue-green (*oöcyan*) eggs are predominantly protoporphyrin-IX and biliverdin-IX, respectively (Lang and Wells, 1987). Shell pigment may be deposited either as a superficial layer, which is generally mottled in appearance, or as a uniform ground-color, which is incorporated throughout the palisade layer of the shell. A significant portion of the pigment in brown eggs of the domestic fowl is in the cuticle (Lang and Wells, 1987).

The proteinaceous cuticular layer (*cuticula*) is deposited over the surface of the egg just prior to oviposition. The stimulus for the uterus to change from the calcification process to the secretion of cuticular material is unknown. The cuticle functions to protect the egg against microbial invasions, and affects the water vapor conductance of egg shells of some but not all species of birds (Deeming, 1987). The cuticle does not affect egg shell strength.

Vagina. With the exception of the sperm-host glands located in the cranial vagina, this segment of the oviduct is devoid of secretory tubular glands. However, ciliated and nonciliated epithelial secretory cells are present, the latter being the source of the acidic polysaccharides found in the vagina.

OVULATION-OVIPOSITION CYCLE

The time interval between oviposition and ovulation, and the time an egg mass spends in each segment of the oviduct has been determined for the gallinaceous species. These studies have shown that the time an egg mass spends in each segment of the oviduct, total time spent in the oviduct, and the interval from an oviposition to a subsequent ovulation is remarkably uniform across species.

Despite these similarities with respect to duration of the ovulation-oviposition cycle, striking differences in egg laying patterns exist. This is because on a 14-hour daily light schedule, the signal for the preovulatory LH release, which is necessary for ovulation to occur, is entrained to the onset of the scotophase (dark phase) in *Gallus*, *Anas*, and probably *Meleagris*, and it is entrained to the onset of the photophase (light phase) in *Coturnix*. Nevertheless, the preovulatory LH release, the so-called open period, appears to be primarily controlled by the bird's circadian system (Phillips et al., 1986). The open period can be advanced by lighting cycles of greater than 24 h and delayed by cycles of less than 24 h. It is only on light cycles of 24 h (14 light:10 dark) that the open period occurs during the scotophase. See Phillips et al. (1986) for a thorough discussion of this topic.

Ovulation. The exact mechanism of ovulation in *Aves* is still questionable. The follicle wall in the region of the stigma (*stigma folliculare*) consists primarily of collagenous tissue from the *theca externa*, and lacks the connective tissue coat found in the remainder of the follicle. Furthermore, the collagenous fibres are oriented parallel to the long axis of the stigma, thereby compromising lateral tensile strength and making the connective tissue fibres relatively easy to separate, as perhaps during ovulation. In regions outside of the stigma the collagenous fibres are randomly interwoven, providing a matrix of greater tensile strength.

Immediately prior to an ovulation, collagenous fibres of the theca externa disassociate into a sparse network, and the underlying granulosa cells (*cellulae strati granulosi*) show evidence of autolytic degeneration. Extruded proteolytic enzymes, acid protease and collagenase, then act to degrade the limited collagen fibres in the region of the stigma, reducing further their finite tensile strength, thereby precipitating a rupturing of the stigma and an ovulation (Ogawa and Goto, 1984).

Plasminogen activator was demonstrated recently in granulosa cells harvested from F_1 follicles of domestic fowl (Tilly and Johnson, 1987). Plasminogen activator functions to convert the pro-enzyme plasminogen to plasmin which, in turn, converts procollagenase into the active enzyme collagenase. In mammals, follicular rupture and ovulation have been associated with plasminogen activator and plasminogen. Because LH, progesterone and testosterone inhibited plasminogen activator activity in hen follicles, and the prostaglandins PGE and PGE_2 stimulated such activity, Tilly and Johnson (1987) speculated that interactions involving these hormones could provide a control mechanism to insure that ovulation is restricted to a precise time interval. Nevertheless, these results are difficult to reconcile with those of Tanaka et al. (1987), obtained with *in vitro* perfused ovaries of domestic fowl. They found that both LH and progesterone precipitated an ovulation four h postinfusion and that the addition of a steroid synthesis inhibitor prevented gonadotropin-induced ovulations. Tanaka et al. (1987) suggested that steroid hormones are essential for the ovulatory process, and that elevated progesterone, possibly the result of LH stimulation, may act directly on the follicle to cause an ovulation.

Oviposition. During an oviposition sequence, contractions by the uterine musculature force the egg through a relaxed uterine sphincter muscle (*musculus sphincter vaginae*) into the vagina, and the resulting distention of the vaginal wall initiates the so-called 'bearing-down reflex' (Sykes, 1955). Characteristics of the reflex include postural adjustments to facilitate oviposition, increased skeletal muscle contraction and increased intra-abdominal pressure. As a result the egg is forced from the vagina through the cloaca and is oviposited. Oviposition can occur in the absence of the bearing-down reflex, but it is delayed. Also, the vagina itself is incapable of expelling an egg.

Neurohypophyseal hormones and oviposition. At the time of oviposition there is an increase in plasma levels of arginine vasotocin (AVT) along with a sharp decrease of AVT in the neurohypophysis. However, the exact role of AVT in oviposition, and stimulus for the release of AVT at oviposition has not been established.

Prostaglandins and oviposition. It is now well established that prostaglandins are intimately involved in the oviposition process, for plasma concentrations of prostaglandins increase significantly at oviposition and drop rapidly immediately thereafter. Also, oviposition can be delayed by passive immunization against the prostaglandin PGE_1, and premature ovipositions can be induced by prostaglandin injections. Furthermore, exogenous prostaglandins are considerably more potent than oxytocin in inducing oviposition, and the pharmacological inhibition of prostaglandin synthesis blocks oviposition (see Hertelendy et al., 1984).

The prostaglandin $PGF_{2\alpha}$ increases muscular contractions and increases pressure in all segments of the oviduct, whereas the prostaglandins PGE_1 and PGE_2 increase muscular contractions in all segments of the oviduct with the exception of the vagina, where they induce a relaxation of the musculature. Both Verma et al. (1976) and Wechsung and Houvenaghel (1976) noted that the divergent responses to the two classes of prostaglandins are an ideal physiological adaptation for the oviposition process. For oviposition to occur there must be an increase in uterine musculature contractility and a concomitant relaxation of both the uterovaginal sphincter muscle and the vaginal musculature, responses that are brought about by the combined action of the PGF and PGE prostaglandins. Thus, as pointed out by Hertelendy et al. (1984), this combined action of $PGF_{2\alpha}$, PGE_1, and PGE_2 may represent the final regulatory pathway of oviposition.

FERTILIZATION

The fusion of male and female gametes (syngamy) represents but one event in a series of closely related preliminary steps or processes. To be capable of fertilizing an ovum, a spermatozoon must have obtained a proper stage of maturity, first in the male reproductive tract and then in the female reproductive tract. Unless properly matured, a spermatozoon cannot undergo an acrosome reaction which allows it to successfully penetrate the yolk membranes. These maturational requirements, however, appear to be considerably less in avian than in mammalian species.

Maturation of spermatozoa. Mammalian spermatozoa undergo subtle physiological and structural changes while residing in the epididymis that are critical to the attainment of functional maturity. Spermatozoa from the rete testes are incapable of fertilization, as are spermatozoa taken from the caput and proximal corpus epididymis. Only spermatozoa taken from the distal corpus epididymis, cauda epididymis and the ductus deferens have the functional maturity to fertilize. The sperm cells also acquire progressively increased motile abilities as they traverse the length of the epididymis. While the acquisition of sustained progressive motility and the ability to fertilize are closely related, the relationship is not absolute. Some fully motile spermatozoa are incapable of fertilizing an ovum.

During passage of mammalian sperm cells through the male reproductive tract, changes occur in sperm membrane net charge and charge density, lectin binding ability, and protein and antigenic composition. The molecular changes that occur in the surface structure of a spermatozoon are prerequisite to developing motile abilities and attaining the capacity to fertilize an ovum.

Spermatozoa from the domestic fowl show similar maturational changes while in the male reproductive tract, but the requirements are not as clearly defined as in the mammal. Howarth (1983) obtained total infertility when spermatozoa harvested from the testes of domestic fowl were inseminated intravaginally, but when spermatozoa taken from the testes, epididymides, or ductus deferentes were inseminated intramagnally, a point in the oviduct where motility is not a limiting factor, high levels of fertility were obtained with spermatozoa from all three sources. The results indicate that, in contrast to mammals, spermatozoa of the domestic fowl attain the ability to fertilize relatively early, possibly at the time of spermiation. The limiting factor with testicular and epididymal spermatozoa when inseminated intravaginally appears to be the absence of progressive motility.

Antibodies developed against fluid secretions of the epididymides from domestic fowl showed that proteins in these secretions normally coat the surface of spermatozoa (Esponda and Bedford, 1985). Morris et al. (1987) obtained similar results, but in addition they found that antibodies developed against chicken epididymal fluid would bind to spermatozoa from turkeys and quail. Nevertheless, the acquisition of sperm binding proteins in the epididymis is apparently not essential for fertilization, because nonbound testicular sperm cells are capable of fertilizing when inseminated intramagnally.

Capacitation of spermatozoa. The physiological change termed capacitation is a prerequisite for mammalian spermatozoa to be able to fertilize an ovum. Intact noncapacitated spermatozoa cannot fertilize an ovum. Capacitation confers upon a sperm cell the competence to initiate an acrosome reaction in response to an appropriate stimulus, resulting in disruption of the acrosome and the release of hydrolytic enzymes. Capacitation develops during exposure to female genital tract fluid, with the necessary time of exposure being dependent upon the species.

Current evidence indicates that capacitation is not a prerequisite for chicken or turkey spermatozoa to fertilize ova *in vitro*. If capacitation is, indeed, required by avian spermatozoa, the reaction must be of an abbreviated duration, for numerous published reports exist indicating that fertilization can occur very soon after natural or artificial insemination. Howarth (1984) suggested that if capacitation is necessary, a maximum 15-min interval is adequate for capacitation to occur in avian sperm cells.

Acrosomal enzymes. Hydrolytic enzymes located in the acrosome region of a sperm cell aid in the penetration of an ovum during fertilization. In mammals a number of acrosomal enzymes have been identified, but only a single enzyme, acrosin, occurs in high levels in avian spermatozoa (see Richardson et al., 1988). The reduced number of hydrolytic enzymes in avian spermatozoa may be a reflection of the fact that only a single investment of limited thickness (2-4 µm) need be penetrated by a spermatozoon during fertilization.

Yolk membranes and fertilization. At the time of ovulation, the wall of the ovum consists of the original, or primary, oocyte cytolemma (*cytolemma ovocyti*) and the thin overlying perivitelline layer (*lamina perivitellina*), termed the inner membrane by Bellairs et al. (1963). The perivitelline layer is assumed to be derived from the granulosa cells of the follicle and as such is considered to be a secondary layer. The perivitelline layer is comprised of a porous (two µm) network of solid fibres, which are thinner in the germinal disc region than in the remainder of the ovum.

After its entry into the infundibulum of the oviduct, two additional tertiary layers are added to the ovum, the continuous layer (*lamina continua*) and the extravitelline layer (*lamina extravitellina*). The continuous and extravitelline layers were called the middle and outer membranes respectively by Bellairs et al. (1963), but the term yolk membranes has since been proposed for the composite membrane (King, 1975).

Based on *a priori* reasoning, it was assumed fertilization occurred in the anterior infundibulum prior to the deposition of the tertiary yolk membranes. However, there is only limited sperm-ovum contact in the anterior infundibulum prior to any membrane deposition. Most contact between spermatozoa and ova is in the posterior infundibulum while tertiary yolk membrane secretion is taking place (Okamura and Nishiyama, 1978a). Because the tertiary yolk membranes possess a loose fibrillar structure during their initial development, the spermatozoa are able to pass through unimpeded to the underlying perivitelline layer. Thus, during the early stages of deposition, the tertiary yolk membranes are not a hindrance to the fertilization process.

Syngamy. The mechanism of fertilization in *Aves* was earlier thought to be a relatively simple procedure because of the reticular nature of the perivitelline layer of the yolk membranes. Enzymatic hydrolysis of the perivitelline layer was thought to be unnecessary, and spermatozoa were perceived as merely passing between the fibres of the porous perivitelline layer (Bellairs et al., 1963). However, the ultrastructural studies of Bakst and Howarth (1977) and Okamura and Nishiyama (1978a) clearly demonstrated that spermatozoa penetrate the perivitelline layer by enzymatically hydrolyzing the underlying fibres, and not by simply passing through the membrane pores.

Penetration of the plasma membrane itself by the spermatozoon involves an initial fusion by the exposed inner acrosomal membrane of the spermatozoon and the plasma membrane of the ovum to form a single continuous membrane. The head of the spermatozoon then enters the ooplasm, the nuclear envelope of the spermatozoon disappears, and the dense sperm chromatin disperses. Later the dispersed sperm chromatin becomes reconstituted, evolving as the male pronucleus enveloped in a dual continuous membrane (Okamura and Nishiyama, 1978b). In domestic fowl chromosome fusion (syngamy) occurs 3-5 h after an ovum enters the oviduct (Bekhtina, 1966; Perry, 1987).

Pathological and physiological polyspermy. In normal fertilization only one sperm cell fuses with the female pronucleus. When multiple spermatozoa fuse with a female pronucleus, a condition known as pathological polyspermy is initiated. If the female pronucleus fuses with two or three spermatozoa, for example, the developing embryo will exhibit a triploid or a tetraploid constitution respectively, conditions which are pathological (Austin, 1965).

In contrast to pathological polyspermy, physiological polyspermy is a normal occurrence in animals with yolk-rich megalecithal ova. The biological advantage is presumed to be an improved chance of syngamy when widely disparate differences in surface area exist between the germinal disc and the total ovum (Austin, 1965). Multiple male pronuclei is a common occurrence in fertilized avian eggs. Bekhtina (1966) found that an average of 60 spermatozoa normally penetrated the germinal disc of domestic fowl during fertilization, but when more than 200 spermatozoa penetrated the disc, abnormal embryonic development resulted.

OVIDUCTAL SPERM TRANSPORT

The mechanism of oviductal sperm transport in domestic fowl differs, depending upon the region of the oviduct. Dead nonmotile spermatozoa or inert carbon particles inseminated cephalic to the uterovaginal junction of an empty oviduct are rapidly dispersed throughout the anterior oviduct, but similar inseminations made into the vagina remain localized. Conversely, viable motile spermatozoa inseminated intravaginally may be subsequently isolated from the infundibulum. Thus motility is a prerequisite for spermatozoa to traverse the vagina, but upon reaching the uterovaginal junction, the cells are transported passively to the anterior oviduct.

Phasic sperm transport. In mammals, oviductal sperm transport is recognized as occurring in sequential phases. An initial phase of rapid sperm transport is followed by a secondary phase consisting of a sustained migration of motile spermatozoa. The phase of rapid sperm transport has been associated with coordinated contractions of the reproductive tract, while the sustained phase appears to rely, in large part, on spermatozoal motility.

Spermatozoa from the rapid phase of transport are thought to be of a relatively inferior quality, for they are generally poorly motile, and a higher percentage of rapidly transported sperm cells are disrupted.

Phasic oviductal sperm transport has not been formally investigated in the avian oviduct, although published data suggest that it does occur. It is well established that natural or artificial insemination into an egg-free oviduct shortly after oviposition usually leads to fertilization of the next released ovum. Because ovulation normally takes place within minutes of oviposition, the implication is that rapid transport of viable spermatozoa exists within the avian oviduct. The data of Howarth (1971) also support the concept of a rapid phase of sperm transport in *Aves*, for he found ^{65}Zn-labelled turkey spermatozoa at the infundibulum within 15 min of an intravaginal insemination. Nevertheless, phasic sperm transport may be confounded, somewhat, in *Aves* by the presence of a magnal egg, which can apparently block sperm transport.

A sustained phase of oviductal sperm transport also exists in domestic fowl and turkeys, for a gradual time-dependent accumulation of radiotracer-labelled spermatozoa in the uterovaginal region has been demonstrated. Also, the uterovaginal sperm storage tubules of hens become increasingly filled with spermatozoa over time.

OVIDUCTAL SPERM STORAGE

Oviductal sperm storage occurs in two regions of the avian oviduct: in the posterior infundibulum immediately adjacent to the magnum (Van Drimmelin, 1946), and at the junction of the uterus and vagina (Fujii and Tamura, 1963; Bobr et al., 1964). The sperm storage tubules located in the infundibulum have generally been referred to as sperm nests and those in the uterovaginal (uv) region have been variably referred to as uv sperm-host glands, uv sperm-storage glands, and uv sperm-storage tubules. The uv sperm-host glands are considered to be the primary oviductal sperm storage site, although the ability of the infundibular sperm nests to retain spermatozoa in a viable state is equal to that of the uv sperm-host glands in domestic fowl (Van Krey et al., 1966), but not in turkeys (Ogasawara and Fuqua, 1972).

Uterovaginal sperm-host gland morphology. The uterovaginal sperm-host glands are tubular invaginations of the oviduct surface mucosa. In domestic fowl, the glands are located in a band of tissue one cm wide on the vaginal side of the junction between the uterus and the vagina. The sperm-host glands, approximately 500 µm in length, are usually simple nonbranched tubules, although branched tubules may be encountered. It has been estimated that up to 24,000 uterovaginal sperm-host glands may exist in the oviduct of the turkey (Goodrich-Smith, 1978).

Ultrastructural studies show that the morphology of these glands is generally similar across species. Although morphology is similar, species differences in secretory potential do exist. Sperm-host glands of all species examined are characterized by a large accumulation of intracellular lipid, but show only limited evidence of protein synthesis. Glandular glycogen synthesis is variable among species with extensive accumulations of intracellular glycogen being common in glands from domestic fowl, but not in glands from turkeys or Japanese quail. Nevertheless, the exact relationship of possible synthetic products within a sperm-host gland to glandular secretion and sperm storage is unknown. Based on the intracellular distribution of cellular organelles, Schuppin et al. (1984) suggested the primary function of the uterovaginal sperm-host glands may be one of absorption and not of secretion.

Infundibular sperm nests. The infundibular sperm nests are morphologically similar to the uterovaginal sperm-host glands. A detailed account of the ultrastructure of the infundibular sperm nests of domestic fowl is provided by Aitken and Johnston (1963).

MOBILITY OF SPERMATOZOA IN THE OVIDUCT

Sperm-oviduct interrelationships. If not viewed in its entirety, oviductal sperm transport and sperm storage in *Aves* may erroneously be perceived to be an extremely inefficient process. The efficiency of sperm transport appears questionable, for 60-90 percent of the sperm cells of an inseminate are lost from the vagina of domestic fowl and turkeys. Furthermore, the sustained continuous release of spermatozoa from the uterovaginal sperm-host glands for transport to the anterior oviduct (Burke and Ogasawara, 1969), occurs even though the oviduct may be blocked by an egg. The efficiency of oviductal sperm storage might also be questioned, for only 40-60 percent of the uterovaginal sperm glands of chickens and turkeys contain spermatozoa after an insemination (Van Krey et al., 1971, 1974), and the maximum capacity of these glands to trap spermatozoa is readily exceeded (Compton and Van Krey, 1979). Despite these seemingly inefficient processes, the fact that following a single insemination the storage capacity of the uterovaginal sperm glands can exceed the functional life span of the stored spermatozoa (Compton et al., 1978) suggests that overall a rather effective system has evolved in *Aves*. Certainly fertility levels in excess of 90 percent for extended periods of time following a single mating or insemination bear this out. A more detailed review of this system follows.

Filling of the uterovaginal sperm-host glands. The time required for spermatozoa to first enter the sperm-host glands following a vaginal insemination is approximately 60-90 min in domestic fowl and turkeys. The glands then continue to fill over time, attaining a maximum level of fullness within 24 h. Isolated sperm-host glands can fill within 15 min when exposed to spermatozoa *in vitro* (Nash et al., 1986), indicating that much of the *in vivo* delay in sperm gland filling is related to sustained sperm transport.

Uterovaginal sperm-host gland filling appears to be affected by oviduct contractility. Uterine contractility reaches peak levels near the time of oviposition in chickens, turkeys and Japanese quail (Shimada and Etches, 1986), and inseminating hens at this time of the egg production cycle has long been known to result in reduced levels of fertility. Nevertheless, when hens were inseminated at or near oviposition, more spermatozoa are retained by the oviduct, and normal levels of fertility are attained if uterine contractility is pharmacologically inhibited (Brillard et al., 1987). Thus, excessive oviductal contractility appears to affect fertility because of an adverse effect on oviductal sperm transport and presumably sperm-host gland filling.

Sperm release from the oviductal storage sites. It was suggested that spermatozoa located in the infundibular sperm nests of domestic fowl are released as a result of distention pressures associated with the transport of an ovum. However, no investigations have been completed to conclusively and precisely delineate sperm release from the infundibular sperm nests.

The mechanism controlling the release of spermatozoa from the uterovaginal sperm-host glands of the domestic fowl is also somewhat vague. Pressures associated with the passage of an egg are not a primary sperm release mechanism (Zavaleta and Ogasawara, 1987). Burke and Ogasawara (1969) reported the presence of spermatozoa in vaginal fluids of domestic fowl to be independent of oviposition, but Bushman et al. (1985) found that events associated with oviposition and/or ovulation influence the rate of sperm release from the sperm-host glands in domestic fowl. In the latter study, sperm release over a protracted period of time was significantly reduced in hens rendered anovulatory through the use of pregnant mare serum gonadotropin.

Compton et al. (1978) found that when genetically labelled spermatozoa were inseminated sequentially into the domestic fowl, the spermatozoa became stratified by genotype within a sperm-host gland, the spermatozoa remained stratified within a gland over time, and emptying of a gland occurred as a sustained leakage of the most superficial sperm cells. These were significant observations, for they demonstrated that the spermatozoa from the last insemination were the first sperm cells to be released for purposes of fertilization. Stratification of spermatozoa within a sperm-host gland explains earlier observations whereby the majority of progeny were derived from the most recent mating or insemination. It was erroneously concluded in the earlier studies that the response was the result of a competitive advantage for the fresher spermatozoa.

Compton et al. (1978) also found that the storage capacity of the sperm-host glands can exceed the functional life span of the stored spermatozoa. These observations taken together have very important genetic and evolutionary implications, for they demonstrate that the great majority of the progeny will be derived from the most recent mating, which in many instances may not be the most dominant or desirable sire.

Van Krey et al. (1981) proposed that sperm cell agglutination may be the basic mechanism controlling the storage and release of spermatozoa from the uterovaginal sperm-host glands. The ability of sperm cells to agglutinate is dependent upon intact plasma membranes. Accordingly, as spermatozoa age and lose cellular integrity, they also lose the ability to agglutinate and are selectively eliminated from a gland. The sustained release of spermatozoa from a sperm-host gland is, therefore, perceived as consisting of a mixture of motile viable and motile nonviable spermatozoa. Such a mechanism is consistent with the concept of a slow leakage (Burke and Ogasawara, 1969) and could also explain why sperm-host glands do not become impacted with dead decaying spermatozoa following repeated matings or inseminations. The simplicity of the mechanism is especially attractive when considering the humoral inconsistencies that exist when ovulation or oviposition occur independently, such as at the beginning or termination of a clutch.

Sperm numbers and fertility. The median volume of a natural copulation by a domestic cock is estimated to be 0.02 ml, a volume which contains approximately 100 million spermatozoa. It is also generally accepted that 100 million spermatozoa per insemination are necessary to maintain high levels of fertility in commercial enterprises, where minimal time is spent with each insemination. Sperm numbers greatly in excess of 100 million spermatozoa per insemination have been recommended, but there is no published evidence to support such a recommendation. Compton and Van Krey (1979) showed that the filling rate, or trapping efficiency, of the uterovaginal sperm-host glands is delimited. Inseminating spermatozoa in excess of a critical number did not significantly increase the rate at which glands filled, indicating that only a finite number of the competing sperm cells are capable of entering a gland during a given period of time.

Artificial insemination of lesser numbers of sperm cells can yield high levels of fertility, although extraordinary care is required to preclude misinseminations. Lake and Ravie (1987) recently reported that inseminating as few as 5.5 million spermatozoa into domestic fowl can yield acceptable fertility providing a diluent with a readily available energy source is employed at the time of insemination.

Sperm retention by the oviduct. An unexpected observation made by Allen and Grigg (1957) is that approximately 90 percent of the ^{32}P-labelled spermatozoa inseminated into the vagina of a domestic hen may be lost via the cloaca. Similar results were obtained in our laboratory with ^{75}Se-labelled sperm cells (R. Reddy, unpublished data), and more recently by Brillard et al. (1987). Howarth (1971) found 83.8 percent of the total radioactivity (^{65}Zn-labelled spermatozoa) inseminated was excreted from the cloaca of the turkey within 30 min of insemination. Thus, only a very small percentage of the sperm cells inseminated contribute to the pool of spermatozoa stored for subsequent fertilization. The results also point out the importance of proper insemination practices.

IMMUNOLOGICAL ASPECTS OF REPRODUCTION

The interrelationships between the immune system and the reproductive system are both intricate and extensive, and a detailed discussion regarding these complex subjects is beyond the scope of this chapter. Discussion will be restricted to relationships between the immune system and female reproduction, primarily oviductal sperm storage and fertility.

It is well established that humoral antibodies to spermatozoa can be induced in female domestic fowl and turkeys by inoculation with semen or a sperm suspension. However, the relationship between humoral antisperm antibody titres and female fertility is equivocal, for while hens immunized against testicular homogenates or spermatozoa may develop high titres of humoral antisperm antibodies, they do not always show a reduction in fertility. Because active immunization has a limited, if any, effect on fertility, doubt has been expressed that low flock fertility can be attributed to immunization during artificial insemination. Utilizing a relatively sensitive enzyme-linked immunosorbant assay (ELISA), Kirk (1988) found no correlation between humoral antisperm antibody titres and fertility in turkey breeder hens.

Local immune response and fertility. Kirk (1988) and Kirk et al. (1988) identified antibody-positive cells and secretions in the uterovaginal sperm-host glands of relatively infertile, but not fertile turkey hens, and suggested that a local immune response may be occurring in poorly fertile hens. Bakst (1987) identified IgG positive cells in the uterovaginal sperm storage epithelium of fertile turkey hens 10-11 weeks after the initiation of egg production. Infertile hens were not tested, so it is conceivable that some hens in that study may have been in the early stages of an immune response.

Seasonal decline in fertility. Natural immunity to spermatozoa has been suggested as being the cause of seasonal trends in fertility in *Aves*. The so-called seasonal decline in fertility, which is a common occurrence in many commercial turkey breeder flocks, has been associated with the immune system. Seasonal declines in fertility are characterized by a gradual drop in fertility over the course of the egg laying period or season, frequently declining 25 percent after approximately 20 weeks of egg production. The decreased flock fertility is not the result of an overall diminished hen fertility, but rather is due to an increasing number of hens becoming totally infertile. The latter is a result of the uterovaginal sperm-host glands being essentially devoid of spermatozoa.

While investigating possible causes of seasonal declines in fertility in turkey breeder hens, Schuppin et al. (1984) noted that the ultrastructure of the sperm-host glands is not affected by the length of time a hen has been producing eggs, and that glands from infertile hens are morphologically indistinct from those of virgin hens or fertile hens. Van Krey et al. (1987) observed intra-epithelial lymphocytes in the uterovaginal sperm-host glands of fertile and infertile hens and proposed that they may function to suppress an immune response, while Bakst (1987) suggested they functioned to sequester sperm antigens or represented an autoimmune response. Van Krey et al. (1987) identified plasma cells within the sperm-host glands of infertile turkey hens experiencing a seasonal decline in fertility, and noted that the presence of plasma cells producing antisperm antibody within an oviductal sperm storage gland could explain the empty glands normally associated with a seasonal decline in fertility. Thus, evidence does exist for seasonal declines in fertility being a local immune response.

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Chapter 4. PRESERVATION OF AVIAN CELLS

Murray R. Bakst

Techniques in organ, tissue, and cell cultures of intact or dispersed avian cells, as well as the storage of avian semen at temperatures above or below freezing, have advanced to where these procedures are routinely performed in most laboratories conducting research in poultry. Possibly due to both the ease of obtaining tissue and the relative success of the procedures, there has been little impetus to freeze somatic cells. Alternatively, the liquid storage and cryopreservation of germ cells, specifically chicken and turkey spermatozoa, are of significant economic importance and consequently have been extensively investigated. See Lake (1986), Lake and Stewart (1978a) and Sexton (1984) for excellent reviews.

Cryopreservation techniques have considerable potential importance to poultry geneticists, particularly those who have the need to preserve isolated or unique cell lines or spermatozoa from specific lines of birds. The primary objective of this chapter is to review the technology presently available in the area of avian cell preservation. Also reviewed are two techniques which have potential application in the area of genetic manipulation of poultry, primordial germ cell isolation and transfer, and embryo culture. Finally, special consideration is given to the problems associated with preserving avian spermatozoa for reasons due to their unique stature with respect to the physiology and economic importance to the breeding sector of the poultry industry.

SOMATIC CELLS

Freezing dispersed avian somatic cells presents no unique problem. Generally, most protocols used for freezing mammalian cells are applicable to avian cells. Although the cryoprotectants glycerol and dimethylsulfoxide (DMSO) are assumed to be equally effective in protecting mammalian cells against freeze-thaw damage, Shannon and Macy (1973) advised that one must determine the best cryoprotectant for a particular cell line. Dougherty (1962) compared the efficacy of glycerol and DMSO using chick embryo fibroblasts (CEF) and noted that the survival rate of frozen cells was clearly better in DMSO than in glycerol. Similar results were obtained by Porterfield and Ashwood-Smith (1962) and Ratnamohan and Spradbrow (1979). The latter authors noted that polyvinyl pyrrolidone and dextran failed to protect avian cells from freeze damage.

Springer et al. (1969) described a procedure for freezing CEF in 10 percent DMSO which resulted in survival rate of more than 90 percent. Briefly, secondary CEF were cultured in M-199 supplemented with 4 percent calf and 1 percent fetal calf serum. When confluency was reached on 60-mm Falcon plastic dishes, the cells were removed by trypsin followed by gentle scraping. After centrifugation to remove the trypsin, the cells were resuspended in the growth medium plus 10 percent DMSO (v/v). The suspensions were transferred to either 5- or 10-ml polypropylene test-tubes and frozen in an air-cooled freezer using 'supercooled' air (-75 to -79° C). To thaw, the tubes were placed directly into a 37° C waterbath. After removal of the DMSO by centrifugation the cells were resuspended in the growth medium and were ready for plating.

McManus et al. (1974) used another method to freeze cells from chicken and duck embryos. Their success with this procedure, which was slightly modified from Springer et al. (1969), indicated that frozen-thawed chicken and duck embryo cells can substitute for fresh cells in plaque neutralization assays.

The cryopreservation of chicken lymphoid cells was described in detail by Stephens and Sharma (1981). Using a programable freezing unit they froze spleen cells and cells from two lymphoblastoid lines in RPM1-1640 medium plus 5 percent fetal calf serum and 5-10 percent DMSO. Although the survival rate of the spleen cells (mean 51 percent) and cells of the lymphoblastoid lines (mean 86 percent) was highly variable, the *in vitro* responses of the frozen-thawed cells to a T-cell mitogen and a cytotoxicity cell assay were comparable to those of fresh cells. These authors concluded that at least these two *in vitro* effector functions were not affected by cryopreservation. With regard to the freezing procedure, Stephens and Sharma (1981) noted that the most critical factors in this procedure were the concentration of the DMSO, the freezing rate, method of removal of the DMSO from thawed cell aliquots, and rate of dilution of cells in the freezing medium.

It is suggested that investigators refer to the American Type Culture Collection (ATCC, 12301 Parklawn Drive, Rockville, Maryland 20852) for information regarding established frozen cultures of various avian cell lines. Upon request, ATCC will provide a catalogue containing technical and procurement information.

PRIMORDIAL GERM CELLS

Techniques describing the isolation and subsequent cryopreservation of primordial germ cells (PGC) for the purpose of preserving specific genetic information either before or after gene insertion have yet to be published. However, the technology for the isolation and both intra- and inter-specific (turkey PGC transfer to chick embryo) transfer of donor chicken embryo PGC to host embryos have been described in detail by Reynaud (1969a, 1969b, 1976) and Shuman (1981). In the realm of genetic engineering, the usefulness of such technology lies in the nature of the PGC. Genetic manipulation of this cell type, possibly through DNA microinjection, and the subsequent transfer and multiplication of the modified PGC in the recipient could result in progeny which express the microinjected DNA (Shuman, 1981).

EMBRYO CULTURE AND CRYOPRESERVATION

Embryo culture. Reviews of early work and problems associated with the use of shell-less (*ex ovo*) approaches to chick embryo culture are found in Silver (1960), Ramsey and Boone (1972), Dunn (1974), and Dunn et al. (1981). Only Richards (1982) has adapted the shell-less culture technique described by Dunn and Boone (1977) to turkey embryos while Ono and Wakasugi (1983) developed an embryo culture system for Japanese quail using a plastic-wrapped surrogate egg.

The shell-less technique has been used to determine the mineral distribution, concentration, and metabolism in chickens (Dunn and Boone, 1976), turkeys (Richards, 1984; Richards et al., 1984), and crosses of wild-type quail (Ono and Wakasugi, 1984) during embryonic growth and development. However, a major drawback to the shell-less culture technique is that the embryos fail to hatch and generally have a mean survival time of 18 days with chick embryos (Dunn and Boone, 1977; Dunn et al., 1981), 20 days with turkey embryos (Richards, 1982), and 14 days with Japanese quail embryos (Ono and

Wakasugi, 1983).

Rowlett and Simkiss (1987) described an embryo culture technique which used a 'surrogate' eggshell taken from eggs of either the same or other species. This procedure resulted in hatched viable chicks. In the development of their surrogate egg procedure, Rowlett and Simkiss (1987) addressed two problems which were felt to be inherent in the shell-less culture system. First, the inefficient absorption of albumen by the embryo, possibly due to the incomplete growth of the extraembryonic membranes, and second, the low levels of blood calcium due to the absence of the shell. Apparently, the proper development of the chorioallantois is critical to the normal long-term growth and development of the chick embryo. Not only is the chorioallantois involved in the absorption of the albumen, but it also functions both in the utilization of the shell as a source of calcium and in the air exchange mechanisms for the respiration of the embryo. The surrogate egg procedure of Rowlett and Simkiss (1987) permits functional, if not normal, differentiation and growth of the chorioallantois either in shells of the same or another species.

Perry (1988) developed another embryo culture system in which the fertilized ovum is first removed from the magnal region of the oviduct and placed in a beaker with a defined medium plus additional liquid albumen. As the embryo develops, the ovum/albumen mass is transferred through a series of separate culture systems until hatching time. With this procedure one is afforded the opportunity to insert exogenous DNA into the germinal disc (blastoderm) with the object of producing transgenic chickens.

Embryo cryopreservation. In contrast to the advanced state of knowledge surrounding frozen embryo technology in mammals, the freezing of avian embryos for the purpose of retrieval of specific genetic lines has not been vigorously investigated. Attempts at short-term maintenance of early embryos (blastoderms consisting of about 500-8,000 cells) excised from the yolk surface generally have not been successful. Olszanska et al. (1984) and Mindur et al. (1985) using Japanese quail and chicken embryos, respectively, transferred blastoderms to filter paper rings and placed them in albumen at an oblique orientation. Few embryos developed beyond the primitive streak stage.

Ratnamohan and Spradbrow (1979) subjected 10-day-old chick embryos to freezing with the objective of maintaining a source of cells for culture work. Each embryo was placed in a vial containing T-199 medium plus 10 percent fetal calf serum and varying concentrations of DMSO, frozen to -70° C, thawed 48 h later, and then subjected to cell dispersal procedures. The authors found that freezing embryos with 25 percent DMSO resulted in a cell survival rate of about 67 percent and a cell yield of about 46 percent, the latter slightly less than half of mean cell yield obtained from fresh embryos.

SEMEN PRESERVATION

In general terms the 'liquid storage' of semen is the practice whereby semen, usually manually collected by the abdominal massage technique first introduced by Burrows and Quinn (1935), is diluted with a buffered salt solution, referred to as an extender or diluent, and is then held at some temperature above freezing until insemination. Cryopreservation involves the freezing and thawing of a semen sample suspended in a diluent containing a cryoprotectant. Since there remains some question as to whether cold shock (also referred to as temperature shock, chilling injury, or thermal shock) is separate from cell injury due to freezing and thawing (Watson, 1981; Mazur, 1985), cold shock and freezing injury will be discussed in separate sections. With respect to avian spermatozoa, Yousif et al. (1984) have suggested that different factors may be responsible for the protection of chicken spermatozoa during liquid storage for 24 h than when frozen and thawed.

Liquid Storage Of Semen

General considerations. The capability to store semen at temperatures above 0° C is crucial to the success of present day artificial insemination (AI) programs in the turkey industry. A current trend in turkey male breeder management is the establishment of centralized male breeder facilities to supply semen for several breeder hen flocks. These centralized facilities are commonly referred to as stud farms.

The stud farm concept became a reality in North America only after turkey semen could successfully be held for at least six h prior to insemination without any loss in fertilizing capacity. The advantages currently realized by the industry include improved male breeder management, increased selection pressure of the male line parent stock, increased ratio of males to females, and the ability to transport diluted semen moderate distances between breeding facilities.

Although semen dilution and storage are now firmly entrenched in AI technology, they have not yet reached their full potential (see Table 4.4). Dilution of semen remains at a rate which translates roughly into a male:female ratio of 1:10, even though semen can be diluted at a rate of 1:20 to 1:30. It is only a matter of time and experience with techniques associated with liquid storage of semen before the full potential of semen extension is realized by the commercial turkey industry. Table 4.1 provides a general outline for the liquid storage of turkey semen.

In contrast to the turkey industry which relies exclusively on AI, the role of AI in the broiler breeder sector is quite limited. Although the same benefits of semen dilution and AI can be realized by the broiler industry, this sector of the poultry industry relies nearly exclusively on natural matings for breeding purposes. Until it becomes economically feasible to do otherwise, AI will remain as an alternative breeder management system for the broiler industry (Bilgili, 1988, personal communication).

Semen evaluation prior to storage. Before processing chicken or turkey spermatozoa for storage semen quality should be assessed. Semen should be discarded if

Table 4.1. A procedure for the short-term (6 h) liquid storage of turkey semen. (Modified from Sexton and Giesen, 1983).

1. Uncontaminated semen (2 ml) is collected into a precooled vial containing 2 ml of diluent held in a thermos (J.E. Edwards Agric-Supply, Inc., Baraboo, Wisconsin) adapted to maintain vials containing semen at a cool constant temperature.
2. The semen is evaluated (sperm concentration, motility, morphology, etc.) at the laboratory and diluted as necessary to get the appropriate concentration of spermatozoa per insemination dose.
3. The diluted semen is transferred to an Erlenmeyer flask to ensure an adequate surface area for aeration of the semen (2-3 ml of diluted semen for a 10 ml flask, 6-8 ml diluted semen for a 25 ml flask).
4. The flask is loosely covered with aluminum foil and secured on a platform shaker in a 5° C waterbath (water level at the level of the semen) or orbital shaker housed in a 5° C cold room for 6 h.
5. When removed from shaker the flask is placed in a cold-water bath and then transported to the hen facility for insemination.

excessive amounts of urates, blood, fecal material, or other debris are present in the sample since such contamination may have a detrimental effect on fertility (Sexton, 1979). Consideration should be given to culling individual males that fail to ejaculate or persistently contaminate ejaculates.

Procedures for the further evaluation of semen are generally performed in the laboratory. Semen volume, sperm concentration, sperm motility, and the percentage of viable and nonviable (dead) spermatozoa are generally examined. Several techniques have been used to estimate the number of spermatozoa in a sample. Counting spermatozoa in a hemocytometer (Sexton, 1976a) is probably the most accurate method and is often used to calibrate and validate other sperm counting procedures. Probably the most popular technique for obtaining an estimate of the sperm concentration is based on the packed cell (spermatozoa) volume as determined with a microhematocrit centrifuge (Jones and Leighton, 1987). Although relatively fast, both the turbidity (Cecil, 1982) and spectrophotometric techniques (Wishart and Palmer, 1986) are not widely used because of the possible interference by particles possibly derived from degenerated spermatozoa.

The percentage of dead spermatozoa is often determined using a vital stain which penetrates and stains dead or damaged spermatozoa. Viable spermatozoa are assumed to have an intact selectively permeable plasma membrane which excludes the stain, while dead spermatozoa, assumed to have a freely permeable plasma membrane, are stained. However, the lack of objectivity and uniformity in reading stained smears of semen samples are the major drawbacks of this procedure. A technique using similar principles of stain exclusion by viable intact spermatozoa has been developed using a fluorescent stain, and the percentage of dead or nonviable spermatozoa is determined objectively using a fluorometer (Bilgili et al., 1985). Regardless of which procedure is used, the usefulness of these stain exclusion techniques as indicators of sperm viability must be questioned when used with turkey semen stored for longer than 18 h since there is an extremely poor correlation between the sperm viability scores and hen fertility (Sexton, 1988).

As with the live-dead stain, sperm motility is also subjectively estimated (Bakst, 1980) and consequently its usefulness in indicating sperm quality may be questioned. Methods have been developed which objectively measure sperm motility. One such procedure relies on the rate of dispersal of spermatozoa in a spectrophotometer flow cell. The rate of change in the optical density of the sample is directly related to the motility of the spermatozoa (Wishart and Palmer, 1986). Another method of objective quantification is the use of a computerized semen analysis system. This system derives information based on sequential digitized images of spermatozoa and provides estimates of the percentage of motile spermatozoa, velocity and path of spermatozoa, and mean lateral head displacement and beat cross frequency. A drawback to this system is that semen samples must be diluted up to 2000-fold to get 20-50 spermatozoa in the examination field. Whether motility values obtained with spermatozoa at such extreme dilutions are representative of the motility of spermatozoa in the original sample has yet to be determined.

Influence of male age and genotype on semen storage. If the semen is to be processed for liquid storage or cryopreservation, consideration should be given to the age of the males at the time of semen collection. Sexton and Giesen (1982), Giesen and Sexton (1983), and Huyghebaert et al. (1985) noted that the viability of turkey spermatozoa subject to 18-24 h of storage decreased as the reproductive season (age of males) progresses. Age of the male at time of semen collection also affects the viability of cryopreserved spermatozoa, as fewer fertile eggs are obtained from hens inseminated with frozen-thawed spermatozoa from 53-week old males compared to 34-week old males (Sexton, 1981).

The tolerance of spermatozoa to liquid storage and cryopreservation also varies between genotypic strains of chickens (Lake, 1983; Bacon et al., 1986; Froman and Bernier, 1987), and between individual males within a population (Lake, 1954). Generally, semen from strains or individuals which demonstrate poor fertility when used fresh, has poor fertility when used after storage. After several generations of male selection based on the duration of fertility of frozen-thawed chicken spermatozoa, Buckland and co-workers (Mitchell et al., 1977; Yousif et al., 1984; Ansah et al., 1985) were able to detect physiological and biochemical differences in spermatozoa between control and selected lines for resistance to cryo-injury. Spermatozoa from the more cryoresistant line had lower levels of cholesterol (Ansah and Buckland, 1982a) and were more permeable to glycerol (Ansah and Buckland, 1982b) than spermatozoa from the control line.

Poultry semen diluents. A diluent is a buffered salt solution used to dilute fresh semen for the purpose of sustaining sperm viability and for maximizing the number of hens that can be inseminated with an ejaculate or pooled ejaculates. Since a universal diluent and accompanying protocol for the liquid storage or cryopreservation of spermatozoa adaptable to all avian species has yet to be devised, diluents and methodology must accommodate species-specific differences, such as sperm membrane composition, metabolic requirements, enzyme systems (acrosomal as well as mitochondrial), and motility.

Furthermore, to optimize fertility the type of diluent and accompanying protocol used for handling semen must correspond to the objectives of the AI program. Referring to Table 4.2, it is apparent that the composition and specific conditions regarding the dilution rate, storage temperature, and the need to aerate by agitation, distinguish the diluents listed. Such conditions represent adjustments to meet the differing metabolic requirements of chicken and turkey spermatozoa and are crucial for maximal survival of chicken and turkey

Table 4.2. Composition and comments on selected diluents[1] used for liquid storage above 0° C and for freezing chicken and turkey semen.

Diluent Component, g	Liquid storage above 0° C[6]					Freezing semen[6]			
	A	B[2]	C	D	E	F	G	H1	H2
Sodium glutamate (monohydrate)	2.01	8.67	0.76	0.96	1.10	8.67	8.70	1.92	1.92
Fructose		5.00				5.00	5.00	0.80	0.60
Glucose	2.01		0.30	0.30	0.36				
BES[3]			1.525		3.05				
TES[4]		1.95				1.95	1.90		
Egg albumen (fresh or freeze-dried), ml	15.0								
Skim milk, ml	20.0								
Polyvinyl pyrrolidone (MW 10,000)								0.30	
Citric acid	0.13								
Magnesium acetate (tetrahydrate)			0.04	0.04	0.08			0.08	0.08
Magnesium chloride (octahydrate)		0.34				0.34	0.30		

Continued ...

Table 4.2. Composition and comments on selected diluents[1] used for liquid storage above 0° C and for freezing chicken and turkey semen. (Continued)

Diluent	Liquid storage above 0° C[6]					Freezing semen[6]			
Component, g	A	B[2]	C	D	E	F	G	H1	H2
Potassium acetate (anhydrous)								0.50	0.51
Potassium citrate		0.64				0.64	0.60		
Potassium citrate (monohydrate)			0.064	0.064	0.128				0.128
Potassium diphosphate (trihydrate)		12.70				12.70	12.70		
Potassium phosphate		0.65				0.65	0.60		
Di-sodium hydrogen phosphate (anhydrous)					0.136				
Sodium acetate (anhydrous)				0.255	0.146				
Sodium acetate (trihydrate)		4.30				4.30	4.30		
Sodium citrate (monohydrate)	0.77								
Sodium hydroxide (1N), ml			2.9		5.6				
Glycerol								13.64	
Dimethylsulfoxide, ml						4%(v/v)[7]	4%(v/v)[8]		
Distilled water to a volume of __ml	100	1000	50	50	100	900	1000	100	100
pH	6.3	7.5	7.05	7.23	7.1	7.5	7.5		
Osmolarity (mosmol/ kg H_2O)	460	333	411	360	402	366		333[5]	

Comments:	A		B[2]	C	D	E	F	G	H1	H2
Species	Chicken	Turkey	Chicken	Chicken	Chicken	Turkey	Turkey	Chicken	Chicken	
Dilution (semen: diluent)	1:1	2:3	1:2	1:1	1:1	1:4	1:4	1:5	1:3	Step-wise addition to thawed semen
Storage time	24 h	6 h	24 h	24 h	5 h	24 h				
Storage temperature	5° C	6-7° C	5° C	5° C	5-15°C	5° C				
Agitation	yes					yes				

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[1] Commercial diluents for turkey semen include IMV (IMV International, L'Aigle, France), MTGA (Minnesota Turkey Growers Association, St. Paul, MN), SemAID (Poultry Health Laboratories, Davis, CA). The composition of these diluents is not available. [2] This is the composition of Beltsville Poultry Semen Extender (BPSE)-I. For the liquid storage of turkey semen at 5-15° C for 6 h BPSE-II is used. BPSE-II has the same composition as BPSE-I except the pH is 6.5 and the osmolarity is 353 mosmol/kg H_2O (Sexton and Giesen, 1982). [3] NN-bis (2-hydroxyethyl) -2-aminoethanesulfonic acid. [4] N-tris (hydroxymethyl) methyl-2-aminoethanesulfonic acid. [5] Without glycerol. [6] A, Van Wambeke (1972), Huyghebaert et al. (1984); B, Sexton (1977); C, Lake and Ravie (1979); D, Lake and Ravie (1980); E, Lake et al. (1984); F, Sexton (1980a); G, Sexton (1976b); H1, freezing diluent, H2, thawing diluent, Lake and Stewart (1978b). [7] Added in bulk to diluted semen after equilibration for 15 min at 10°C. [8] Added in bulk to diluted semen after equilibration for 2 h at 5°C.

spermatozoa. This concept is further highlighted by Lake and Wishart (1984) in which they discuss physiological differences between chicken and turkey spermatozoa and the implications such differences have with regard to semen storage technology.

Diluent composition and pH. A diluent is generally composed of a buffering system, a chelator, an energy source, and a balanced mixture of salts. Glutamate is a component common to most poultry semen diluents. It is the major nonprotein nitrogen constituent and the major anion in seminal plasma of chicken and turkey semen. Although the biological significance of glutamate *in vivo* is not known, it is thought to help maintain the osmotic pressure of semen and/or serve as a chelator (Sexton and Fewlass, 1978). It is doubtful that glutamate is used by spermatozoa as an energy source since the glutamate dehydrogenase activity in chicken semen is low (Buckland, 1970). Nevertheless, the importance of glutamate to overall diluent composition has been demonstrated, for the absence of glutamate from the Beltsville Poultry Semen Extender significantly reduced the viability of chicken spermatozoa (Sexton and Fewlass, 1978).

The buffering system of the diluent, which could include a mixture of phosphates, citrates, and/or organic buffers such as TES and BES (see Footnotes, Table 4.2 for chemical names) must maintain the desired pH during semen storage. Diluent pH influences both motility and metabolic rate of spermatozoa. Bogdonoff and Shaffner (1954) and Pinto et al. (1984) found that chicken and turkey spermatozoa in high pH media exhibited high metabolic rates *in vitro.* Pinto et al. (1984) cautioned that this relationship (high pH leading to a higher metabolism) does not necessarily mean increased fertility. To the contrary, Sexton and Giesen (1982) noted that turkey semen diluted in Beltsville Poultry Semen Extender at pH 6.5, when compared to the same diluent at pH 7.5, had lower sperm motility but higher sustained fertility after storage for 6 h at 15° C.

Diluent osmotic pressure. There is considerable disagreement regarding proper diluent osmolarity. Diluents ranging from about 250 to 460 mosmol/kg H_20 have resulted in good fertility in both chickens and turkeys (Table 4.2). Tsukunaga and Takahashi (1961) and Yamane et al. (1962) advocate diluents with osmolarities of 484 and 554 mosmol/kg H_20, respectively, to prevent the sperm defect referred to as a 'neck-bent' or 'bent' spermatozoa.

Bent spermatozoa, which are characterized by an acute flexion in the region of the midpiece such that the head is nearly parallel to the tail, are often highly motile, but their presence is negatively correlated with fertility (Saeki, 1960). This is probably the most common defect found in diluted and stored chicken and turkey spermatozoa and appears to be due to changes in the osmotic environment (Van Wambeke, 1977; Bakst and Sexton, 1979; Bakst, 1980; Tsukunaga, 1981).

Osmolarity of the diluent and the osmolar conditions associated with each step in the progression leading to the freezing and thawing of spermatozoa are critical since changes in osmotic pressure associated with each step have been implicated as the source of morphological damage to chicken and turkey spermatozoa. Adverse osmotic conditions resulting from the addition of diluent to fresh semen was suggested as the primary factor leading to alterations in the plasma membrane of chicken spermatozoa (Bakst and Sexton, 1979) and turkey spermatozoa (Bakst, 1980). Furthermore, subjecting chicken spermatozoa (Tsukunaga, 1981) and turkey spermatozoa (Bakst, 1980) to hypotonic solutions resulted in sperm anomalies similar to those observed during freezing and thawing, suggesting that spermatozoa cannot tolerate extreme changes in osmolarity.

Cold shock. By definition cold shock for spermatozoa is manifested by the loss of motility and fertilizing capacity when the semen is cooled rapidly to a temperature above freezing (Watson, 1981). Wales and White (1959) concluded that chicken spermatozoa are quite resistant to such stress based on motility estimates and the percentage of dead spermatozoa following cooling. Similar conclusions can be drawn from other work which shows that the motility of both chicken and turkey spermatozoa is not affected when semen is cooled slowly to 5° C (Bakst and Sexton, 1979; Sexton, 1981). Finally, given the exceptionally high fertility of chicken and turkey hens after insemination with semen stored for 18-48 h at 2-7° C (Wishart, 1981; Giesen and Sexton, 1983; Huyghebaert et al., 1984; Lake et al., 1984), one could conclude that chicken and turkey spermatozoa are not susceptible to cold shock. However, if spermatozoa, particularly after 18 h storage, are subjected to critical assessments using objective techniques and methodologies to obtain the percentage of viable and motile spermatozoa, the new data may reveal that like most cells a substantial number of chicken and turkey spermatozoa suffer from cold shock.

As noted previously in this chapter, the procedures for estimating the number of dead spermatozoa and the percentage of motile spermatozoa are imprecise. Even the procedure for estimating the relative fertilizing capacity of fresh diluted spermatozoa versus cooled stored spermatozoa can be misleading. Inseminating groups of hens with 100 million or more spermatozoa and determining the percentage of fertile eggs laid after each insemination is a nonquantitative measure of the fertilizing capacity of the spermatozoa being examined (Wishart, 1985). Inseminating a fixed number of spermatozoa can highlight differences in semen quality between treatments, but quantitative estimates of these differences are lost when the number of spermatozoa inseminated is in excess of that necessary to obtain maximum fertility. Wishart (1985) proposed inseminating hens with a range of sperm numbers, evaluating the fertilizing capacity of the spermatozoa within a selected dose-range, and then expressing this relationship as the number of spermatozoa within a treatment necessary to fertilize a certain percentage of eggs (see Figure 4.1). Only when objective quantitative semen evaluation procedures are used can a determination be made as to whether or not chicken and turkey spermatozoa suffer from cold shock.

Sperm lysis during storage. Assuming chicken and turkey spermatozoa are subject to cold shock, then the distinction between damage due to adverse osmotic conditions and damage due to cold shock may be difficult to delineate. To further complicate the situation another anomalous condition observed in turkey semen stored at 5° C and 15° C for 18 h is sperm lysis (Giesen and Sexton, 1983; Sexton, 1987, 1988). Whether or not sperm lysis is a manifestation of sperm degeneration (Giesen and Sexton, 1983) has not yet been determined. Nevertheless, sperm lysis is a phenomenon which must be considered, for the total number of spermatozoa recovered at the end of a storage period is less than that of the sample immediately after dilution. The rate of turkey sperm lysis ranges between 17 and 33 percent of the unstored controls (Sexton, 1988). Consideration must also be given to the season during which males are producing semen since semen collected in the spring and summer is more susceptible to sperm lysis than semen collected in the fall and winter (Sexton, 1987).

Relationship between diluents, sperm structure and fertility. Sexton (1988) noted that even when all hens were inseminated with the same number of viable spermatozoa, there remained differences in fertility (sperm viability) between the diluents. He suggested that factors other than the integrity of the sperm plasma membrane were affecting sperm viability. One possibility is that the semen sample is comprised of a

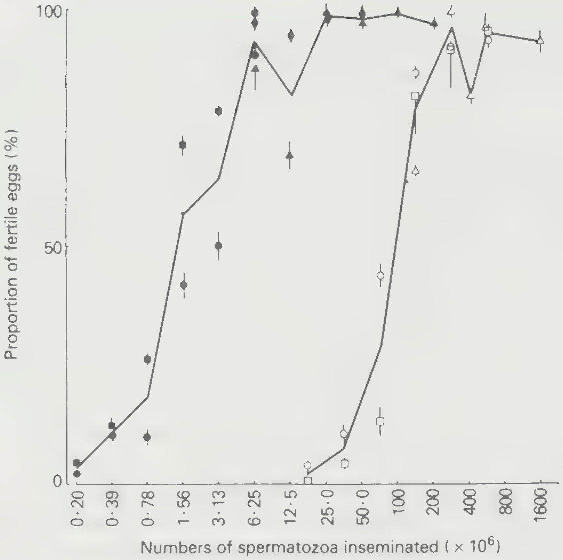

Figure 4.1. The effect of different numbers of fresh and of frozen and thawed chicken spermatozoa on hen fertility during days 2 to 8 following insemination. Each point represents the mean percentage ± SE of fertile eggs laid by groups of 8 hens. Eggs fertilized by fresh and frozen semen are represented by closed and open symbols, respectively. Different shaped symbols (open or closed) represent different insemination experiments. To obtain 50 percent fertility, 1.56 and 100 million spermatozoa (a 64-fold difference) must be inseminated from the fresh and frozen semen, respectively. Yet, fertility levels greater than 90 percent for both semen samples are achieved when 150 million or more spermatozoa are inseminated. (From Wishart, 1985).

heterogeneous population of spermatozoa, each subpopulation endowed with some degree of resistance to the stress of dilution, cold shock, and changes in osmolarity. Different diluents and their associated protocols may actually select for or against one or more of these subpopulations.

Additional factors may also influence the morphology and subsequent viability of spermatozoa at the time of dilution and storage. Damage due to short-term (2 h at 5° C) liquid storage of both chicken and turkey spermatozoa is similar and includes distention of the plasma membrane, bent spermatozoa, and to a lesser extent swelling of the mitochondria (Bakst and Sexton, 1979). Such damage may be a manifestation of cold shock. Hypoxic injury, observed in some somatic cells *in vitro*, is likewise characterized by alterations of the cell plasma membrane, including blebbing and increased membrane permeability (Herman et al., 1988). Considering that the work with chicken and turkey spermatozoa cited above was completed before the practice of agitating the semen during liquid storage (Wishart, 1981) one must question whether the sperm anomalies observed during storage were due to adverse osmotic conditions alone or the result of hypoxic conditions in the diluent or a combination of both.

Regardless of the basis of this damage, the sperm plasma membrane, particularly around the midpiece, appears to be the organelle of least resistance to stress (Yamane et al., 1966). Stabilization of the plasma membrane may lead to better *in vivo* sperm survival following *in vitro* storage.

Dilution effect. The phenomenon known as the dilution effect can be defined briefly as a short-term increase in sperm respiration and motility followed by a loss of viability after diluting semen with an appropriate diluent (Mann, 1964). Dilution effect appears to be dependent upon several factors, including the type of diluent, rate of dilution (expressed as a ratio of semen to diluent), and the investigator's capacity to discern differences in fertility due to either "the harmful effects of any component of a given diluent" or the "dilution effect" (Sexton, 1976a). Sexton (1987) further suggested that to study this dilution effect one must also examine the diluents, the accompanying methodology, and even take into consideration the time of year when males are producing semen. Nevertheless, dilution *per se* (not exceeding 4 parts diluent to 1 part semen) does not appear to affect chicken and turkey spermatozoa given the vast success of AI programs using diluted unstored semen. Even extreme dilution rates have been successful. Lake and Ravie (1987) obtained 92 percent fertility with chickens after inseminating 5.45 million spermatozoa diluted 46-fold with *ductus deferens* seminal plasma. The combination of a high dilution rate using seminal plasma from the *ductus deferens* and low sperm number suggests that the classic dilution effect may, in fact, be due to the absence of some components in the diluent which are present in seminal plasma.

Cryopreservation Of Semen

General considerations. The body of literature available in the area of cryobiology is varied and extensive. Detailed treatments addressing theories of the basis of cryoprotection and cryoinjury of animal and plant cells can be found in works by Meryman (1966), Smith (1970), Ashwood-Smith and Farrant (1980), and Grout and Morris (1987). In addition, a limited number of reviews relating the principles of cryopreservation specifically to the preservation of mammalian spermatozoa (Johnson and Larsson, 1985; Hafez, 1987; Amann and Pickett, 1987) and fish spermatozoa (Stoss, 1983) are available. Since it is not the purpose of this section to present a detailed account of such information, only a brief overview of the basic concepts of cryopreservation with emphasis on spermatozoa will follow.

Freezing injury. Certain physical changes transpire within and between cells during freezing and thawing which, in the absence of a cryoprotectant, ultimately can be lethal to the cells. In addition to cold shock, intracellular and extracellular ice formation, cell dehydration, and distortion of the organelles and membranes have been cited as potential sources of injury to spermatozoa during freezing and thawing (Amann and Pickett, 1987).

In the following paragraphs general principles regarding the behavior of cells during freezing are described. None has been demonstrated with avian spermatozoa. For the purpose of better understanding the fate of avian spermatozoa during freezing it is assumed that avian spermatozoa in diluent behave in a similar manner.

When cells in a suspension are cooled to -5° C, both the intracellular and extracellular water remain in an unfrozen 'supercooled' state. Ice crystals appear in the extracellular water as the temperature drops below -5° C, while the intracellular water remains supercooled (Mazur, 1985). With progressive lowering of the temperature extracellular ice crystals continue to deplete the fraction of supercooled water now restricted to narrow channels between the extracellular ice crystals (Courtens and Paquignon, 1985; Mazur, 1985). The continued loss of this extracellular water increases the concentration of solutes in the remaining supercooled extracellular water, thus creating a disequilibrium between the extracellular and intracellular compartments. Equilibrium is re-established by osmosis; water moves out of the cells into the extracellular channels.

It is the rate of movement of this intracellular water out of the cells which is crucial to the subsequent fate of the cells. If cooling is too rapid, the cells are not able to lose water fast enough and intracellular ice crystals are formed. Intracellular ice crystals appear to disrupt the integrity of the cells and can be lethal (Meryman, 1971), whereas extracellular ice crystals may not be as damaging since the cell plasma membrane appears to prevent growth of the ice crystal into the cell (Amann and Pickett, 1987). However, if cooling is too slow the cells are excessively dehydrated and consequently the intracellular solutes reach a concentration which may damage the spermatozoa. Therefore, one must find the optimal rate of cooling which minimizes both the detrimental effect of high solute concentration during slow freezing and intracellular ice crystal formation during fast freezing. More detailed analyses of the fate of intracellular water during freezing and how to estimate the probability of intracellular freezing, which is highly correlated with cell death, as a function of cooling rate, can be found elsewhere (Mazur, 1985).

Thawing. With respect to thawing, generally the rate of thawing should approximate the rate of freezing. If the freezing rate was extremely rapid, a procedure which results in small and less deleterious ice crystals, then thawing also should be very rapid to prevent the formation of large intracellular ice crystals, which can be deleterious to cells (Amann and Pickett, 1987). However, if cells which were frozen at a rate slow enough to avoid intracellular ice are rapidly warmed, they may be subjected to osmotic shock (Mazur, 1985). Although equilibration between the intracellular and extracellular compartment is established during slow freezing by osmosis, equilibration may also be reached by the influx of solutes into the cell during slow freezing. Such cells may be subjected to osmotic damage if the thawing rate is too fast to permit the diffusion of the excess solutes out of the cell (Mazur, 1985).

Cryoprotective agents. Cryoprotective agents are compounds which when added to the semen extender can reduce to some degree the detrimental effects of freezing. By penetrating into the cell, a cryoprotectant lowers the temperature at which the supercooled intracellular water initiates ice crystal formation. Cryoprotectants also reduce the effects of slow cooling by preventing excessive concentrating of the extracellular solutes. Whether the increase in extracellular solute concentration *per se* is in fact inimical to cells has recently been questioned. Recent evidence (Mazur, 1985) indicates that, at least for erythrocytes, it is not the gradual rise in extracellular solute concentration during slow freezing which is detrimental to the cells, but instead it is the decrease in the fraction of unfrozen water in the extracellular compartment. If more than 85 percent of the extracellular water freezes, cell survival is low (Amann and Pickett, 1987). A cryoprotectant plus the solutes normally found in a diluent would, at any given temperature during cooling, have a lower concentration of solutes and a greater percentage of unfrozen water than the same diluent in the absence of cryoprotectant. Thus, it is assumed that with a cryoprotectant, spermatozoa in the unfrozen extracellular water channels have more time for intracellular water to move to the extracellular compartment (Amann and Pickett, 1987).

The minimum concentration of cryoprotectant which offers maximum protection must be found with each cell type since most cryoprotectants at a certain concentration can be toxic to cells, particularly spermatozoa. More detailed analyses of the action of cryoprotectants can be found in Fahy (1986) and Meryman (1971).

Cryopreservation of avian spermatozoa. Cryopreservation of avian spermatozoa was first attempted by Shaffner et al. (1941) and Shaffner (1942). A major advance in modern cryobiology was the discovery that chicken spermatozoa frozen in the presence of glycerol still retained their motility after thawing (Polge, 1951).

The cryopreservation of chicken and turkey semen has advanced to where commercial breeders can maintain frozen stores of grandparent breeder lines. Whether secondary breeders (producers of commercial hatching eggs) will ever devote a significant portion of their breeding program to the use of frozen semen is questionable for several reasons, not the least of which is the absolute volume of semen necessary to satisfy the insemination needs of this segment of the poultry industry. At least for the turkey industry, not until AI technology has progressed to where a single insemination of thawed semen results in sustained high fertility for 2-3 weeks, rather than one week or less, can breeders begin to consider the large scale application of frozen semen on commercial farms. A more realistic benefit of the ability to freeze semen successfully is the relative ease and low cost of shipping such genetic material anywhere in the world. A more thorough treatment of the possible applications of a frozen semen program by a breeding operation is presented by Lake (1986).

Outside the commercial poultry industry there is considerable interest by research and zoological institutions in the application of semen cryopreservation in order to maintain both gene pools from birds of low fecundity and the genetic diversity in rare and endangered species of birds. It was found that if frozen-thawed semen from a selected line of inbred chickens known for their low fecundity was surgically inseminated into the magnum, the resulting fertility was high enough to warrant preserving the germplasm by freezing rather than continued propagation (Bacon et al., 1986). Inseminations of frozen-thawed semen

Table 4.3. A procedure[1] for the freezing and thawing of chicken spermatozoa. (Modified from Lake and Stewart, 1978b, and Lake et al., 1981).

Freezing:

1. Uncontaminated semen (.15 ml) is collected in thin-walled (75 x 10 mm) glass tubes and transported to a 5° C cold room within 15 min of collection.
2. After 3 min of temperature equilibration the glycerolyzed diluent (.45 ml, see Table 4.2) is added to the semen, gently mixed, and transferred to a 1 ml ampule.
3. The ampules are sealed, placed on canes, and immersed in a cold alcohol bath for transfer to a programable minifreezer.
4. The glycerolyzed semen is cooled at a rate of 1° C/min to -35° C.
5. After 5 min at -35° C the cane is manually plunged into liquid nitrogen for storage.

Thawing and glycerol removal:

1. The canes are removed from the liquid nitrogen and agitated in a cooled (2-5° C) alcohol bath.
2. The thawed semen is then transferred to a 10 ml conical centrifuge tube immersed in a 2-5° C water bath.
3. At 3-5 min intervals the following volumes of cold nonglycerolyzed diluent (see Table 4.2) are added to the thawed semen: 0.08, 0.22, 0.40, 0.73, 1.5, and 1.9 ml.
4. The diluted semen sample is centrifuged at 700g for 15 min at 5° C and the supernatant is removed. Spermatozoa are resuspended in 0.1 ml of the fresh nonglycerolyzed diluent.
5. The reconstituted semen is transported in a cold-water bath to hen house.
6. Within 20 min of thawing, semen (about 0.1 ml) is inseminated in the vicinity of the uterovaginal junction (deep insemination). Steps 1-6 above are to be repeated at 3-day intervals for 10 days.

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[1] All steps are either conducted in a 5° C cold room or with vials kept in a 2-5° C water bath. All equipment and solutions should be precooled to 2-5° C.

from wild birds has been successful and resulted in progeny from Aleutian Canada geese, American kestrels, and several species of cranes (Gee, 1986).

The development of a protocol for the cryopreservation of avian spermatozoa is quite demanding. Retrieval of a somatic cell line, even with relatively few viable cells after thawing, is possible if when the cells are cultured there is successful regrowth and cell division, an alternative not possible with spermatozoa. Furthermore, unlike mammalian spermatozoa which fertilize single or multiple ova normally within 24 h or less after insemination, avian spermatozoa must survive for several days to several weeks in the oviductal sperm-storage tubules in order to successfully fertilize a daily succession of ovulated ova.

By commercial industry standards there has been limited success in the cryopreservation of chicken and turkey spermatozoa. But by using a freezing and AI protocol described by Lake and Stewart (1978b), cryopreservation of chicken spermatozoa (summarized in Table 4.3) is feasible. Considerations such as precooling all equipment and solutions and beginning the freezing procedure no longer than 15 min after semen collection may be partly responsible for the success of this procedure. Their success was also probably

Table 4.4. Chicken and turkey hen fertility after storage of semen.

Reference and semen treatment	Fertility and time after insemination	Placement and frequency of insemination	Number of spermatozoa or volume of semen
Chicken:			
Van Wambeke (1972) 24 h liquid storage	93% days 2-8	midvagina once only	0.1 ml
Lake and Ravie (1979) 24 h liquid storage	87% days 2-8	- once only	0.6 ml (120-180 x 10^6)
Sexton (1980b) frozen-thawed	61% days 3-7	vagina 2 consecutive days	
Lake and Stewart (1978b) frozen-thawed	80% days 2-6	deep vagina once only	0.1 ml
Bacon et al. (1986) frozen-thawed	53% days 1-6	intramagnal once only	0.1 ml
Turkey:			
Lake et al. (1984) 24 h liquid storage	92% 14 weeks	midvagina[1] weekly	60-180 x 10^6 [1]
Sexton et al. (1984) 6 h liquid storage	87% breeding season	vagina[2] weekly	0.3 ml
Sexton (1980b) frozen-thawed	50% 4 weeks	vagina 5-day intervals	200 x 10^6

[1] Hens were inseminated twice during the week prior to the expected onset of egg production and thereafter weekly with progressively increasing numbers of spermatozoa.

[2] Hens were inseminated three times during the week prior to the expected onset of egg production. This study was a field trial involving six commercial breeder flocks.

augmented by using only males that produced high quality semen and at insemination, depositing the frozen-thawed semen in the vicinity of the uterovaginal junction sperm-storage tubules (refer to Chapter 3 for discussion on oviductal sperm storage). Fertility levels obtained using this protocol are shown in Table 4.4.

The step-wise dilution of the thawed glycerolyzed semen with nonglycerolyzed diluent (Lake and Stewart, 1978b) probably affords a gradual reduction in the intracellular concentration of glycerol thereby minimizing excessive osmotic gradients which can be created during rapid removal of the glycerol. It is these osmotic gradients which are likely responsible for some of the damage conferred to spermatozoa during freezing and thawing.

Despite the undisputed fact (Lake et al., 1980) that the glycerol concentration in thawed semen must be lowered about 1.1M (the concentration necessary for cryoprotective action) to 0.163M or less (the maximum concentration at which glycerol does not significantly reduce sperm fertility), glycerol remains the cryoprotective agent of choice by most investigators. Alternatively DMSO has been shown to be nontoxic to spermatozoa and does not need to be removed from the semen prior to insemination (Sexton, 1976b, 1980a, 1980b; Lake and Ravie, 1984).

Coupling of the cryoprotective agent to the appropriate diluent is critical. By withdrawing individual components of the diluent and inseminating hens with semen diluted with the complete or incomplete Beltsville Poultry Semen Extender (BPSE) with and without 4 percent DMSO (v/v), Sexton (1980a) noted that omission of specific components of the BPSE may have a detrimental effect on fertility only if DMSO was present. He concluded that the cytotoxicity of DMSO is directly related to the composition of the diluent which with it is mixed.

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Part II. QUALITATIVE GENETICS

Chapter 5. GENETICS OF PLUMAGE, SKIN AND EYE PIGMENTATION IN CHICKENS

J. Robert Smyth, Jr.

The seemingly infinite variations in plumage, skin and eye color of the domestic fowl have had an aesthetic appeal to man since the first mutant phenotypes appeared among early domesticated stocks. While many poultry fanciers still breed for the beauty and technical excellence of coloration as set forth by agreed standards, pigmentation is also of economic importance to the commercial poultry industry. Such genetically determined choices as yellow vs. white skin are useful to satisfy the preferences of specific geographic markets, as is the choice between colored and white plumage. Specific color crosses are useful for sex determination of day-old chicks, and more recently, as a model for gene action at the cellular level (Brumbaugh et al., 1979; Wilkins et al., 1982; Boissy et al., 1987). Beyond this, pigmentation in the chicken has proven of value in the important field of developmental genetics starting with the classic embryonic skin exchange studies of Eastlick (1939), Dorris (1939) and Willier and Rawles (1940). An inherent pigment defect has also been shown to be a valuable model for autoimmune disease resembling human vitiligo and an associated retinal dystrophy (Smyth et al., 1981; Lamont et al., 1982; Boissy et al., 1983). This combination of beauty and utility makes the genetics of cutaneous and ocular pigmentation particularly interesting.

Poultry geneticists generally subscribe to the use of a wild-type standard as a basis for gene symbolism, as suggested earlier by Kimball (1951), Jaap and Hollander (1954) and Morejohn (1955). They designated wild-type alleles by a "$^+$" superscript. Although there is no convincing evidence to identify the true ancestry of modern domestic fowl, of the four possible junglefowl candidates the red junglefowl (*Gallus gallus*), has been generally accepted for this role (see Chapter 1). *Gallus gallus* features the sexually dichromatic black-breasted red feather pattern and has melanized slate-colored scales on its shanks. For practical reasons such as excellence in livability and productivity, the Light Brown Leghorn (*Gallus domesticus*) has also been used as a representative for wild-type as suggested by Smyth (1965). Like *Gallus gallus*, it has the black-breasted red feather pattern but differs by the absence of dermal melanin in the shanks and by the ability to deposit yellow carotenoid pigments in cutaneous tissue.

THE INTEGUMENTAL AND OCULAR PIGMENTS

Pigments of the chicken include two general types, the melanins and the carotenoids. Melanins are responsible for feather coloration and the dark pigments of the skin and connective tissue, while the carotenoid, xanthophyll, gives the yellow coloration to the skin, fat and egg yolk. The two types can also interact with each other, and/or other cell types, to produce a variety of shank and eye colors, as well as structural sheens in the plumage.

Melanins

Melanins represent one of the most widespread classes of natural pigments in living organisms, and play a major role in the coloration of feathers, skin, shanks, beak and eyes of chickens. Melanins are divided into two main types, eumelanin and pheomelanin, based on differences in color, chemical composition, solubility properties and associated pigment granule structure. Eumelanin is the pigment of black or blue feathers, the eye, skin, and connective tissue, while pheomelanins appear to be deposited only in red-brown, salmon and buff-colored feathers. Melanin polymers are attached to a protein matrix called a premelanosome. Following melanization, these membrane-bound cytoplasmic organelles are referred to as melanosomes or pigment granules (Searle, 1968; Brumbaugh, 1968). Under normal conditions, melanin in the fowl occurs only as a part of these melanoprotein organelles.

Melanins appeared early in evolutionary history and still exist basically unaltered in present-day birds and mammals (Prota, 1980). Melanins of the feathers, skin and eyes arise from the oxidation of the phenolic amino acid tyrosine by a copper-dependent enzyme, tyrosinase, to form dopa (3, 4-dihydroxphenylalanine) which is further oxidized by the same enzyme to dopaquinone. Through a different enzyme system, dopa also serves as the precursor of dopamine, and ultimately such important products of neural origin as norepinephrine, epinephrine and the neuromelanins, which are chemically analogous to eumelanin (Prota, 1981). All non-neuromelanins share the same tyrosine→dopa→ dopaquinone pathway after which the eumelanin and pheomelanin pathways separate.

Eumelanin synthesis in the chicken appears to follow the classic Mason-Raper pathway involving a series of spontaneous reactions (cyclizations and oxidations) resulting in the eumelanin polymer. However, more recent studies of mammalian hair pigmentation indicate that there are several additional factors involved in the regulation of melanin synthesis (Prota, 1980; Körner and Pawelek, 1982). Pheomelanin formation involves the addition of sulfhydryl (-SH) groups to dopaquinone to form cysteinyldopa. This is followed by several oxidative reactions to yield pheomelanin. Although the eumelanin-pheomelanin switch can also be accomplished using glutathione, cysteine is the more likely instigator, and its levels are high in red chicken feathers (Prota, 1980).

Red and yellow chicken feathers have also been shown to contain pheomelanin-related pigments called trichochromes (Smyth et al., 1951; Prota, 1980). Trichochromes have a low molecular weight and are readily extracted from feathers with mineral acid. Red to red-purple in acid solutions, they become yellow to peach-colored at basic pH's, according to Smyth et al. (1951). They found two trichochromes in red and buff chicken feathers, one of which appeared to be identical to a pigment found in human red hair by Flesch and Rothman (1945). Since feather color does not change following extraction of the trichochromes, their role in feather color is not clear. However, only one of the two chicken trichochromes, and this in reduced quantities, was found in the salmon-colored feathers of female Brown Leghorns and wheaten hens. Both are absent from the brownish body feathers of wild-type (e^{+}) and brown (e^{b}) females. Subsequently, additional trichochromes were isolated from red chicken feathers by Prota and Nicolaus (1967), and Prota (1972). At least some of these appear to be artifactual, since they have not been found in biological material (Prota and Nicolaus, 1967; Rorsman et al., 1977). In any event, trichochrome presence and quantity is related to feather color, and therefore, indicative of genetic differences among certain plumage colors.

In mammals, mixed melanin polymers containing both eumelanin and pheomelanin occur and give rise to a range of intermediate types, probably explaining in part the wide range of hair colors (Prota, 1981). There appears to be no evidence that this occurs in the chicken system where the pigment appears to be either eumelanin or pheomelanin. If such mixed melanins do occur in the chicken, the author suggests the brown-colored feathers of the female Brown Leghorn as a possible source.

Melanin is produced by specialized cells of ectodermal origin called melanocytes. Embryologically melanocytes arise from one of two sources. The retinal and iridial pigment epithelia, and probably the pigment cells of the ocular pecten (Hodges, 1974), arise from the outer layer of the optic cup, while those populating other body sites, including the uveal tract and anterior surface of the iris, originate in the neural crest. It has been generally assumed that a single type of neural crest cell serves as the source for melanocytes found in a variety of pigmented tissues, their differences in form and activity resulting from cyto-environmental interactions (see LeDouarin, 1982 for review). In contrast, Boissy (1986) has observed two separate types of pigment cells present in neural crest cell cultures. One kind produced pigment granules like those found in melanocytes of regenerating feathers, while the other contained melanosomes resembling those of the choroid.

Migration of the undifferentiated melanoblasts starts during early embryogenesis. Embryonic grafting experiments have shown that they have reached such sites as the limb bud at 80-91 h incubation (Watterson, 1942). Shortly after reaching their destination, those destined to produce melanin differentiate into melanocytes characteristic for the particular tissue in which they reside. Some types, like those of the uveal tract of the eye, those found in connective tissue membranes and fascia, around blood vessels, and among the interstitial cells of the testis, appear to differentiate, produce pigment, and thereafter, remain melanogenically inactive. Their melanosomes remain internalized and are not transferred to other cell types as are those of epidermal structures such as the feather. The process of feather pigmentation also differs in that as migrating melanoblasts reach those areas that will give rise to feathers, they become grouped into dermal reservoirs of undifferentiated cells that are in part activated by unknown stimuli to populate each new regenerating feather (Foulks, 1943; LeDouarin, 1982).

Migration from the melanoblast reservoir into a developing feather appears to follow along blood vessels into the dermal papillae and the dermal pulp (Rawles, 1944). They proceed to enter the epithelium at the base of the ramogenic zone, an area organized as a collar of epithelial cells around the vasular pulp. Differentiated melanocytes may already have started to produce pigment. As shown in Figure 5.1, melanocytes then line up adjacent to the interface between the barb ridge and the pulp and become dendritic (see Lucas and Stettenheim, 1972 for review). Melanosomes are produced in the cytoplasm of the perikaryon and translocated through the melanocytic dendrites to the region of the developing columns of barbule cells or keratinocytes. Pigment granule transfer occurs when the tips of the dendrites are phagocitized by the keratinocytes. As the feather grows, the barbule cells become keratinized, while the accompanying melanocytes die and become incorporated into the feather structure (see Bowers, 1988 for review). Therefore, the melanocytes that migrate into a developing feather germ exist and function only during the formation of that feather.

Melanosomes are produced by the coordinated efforts of two separate genetically controlled pathways. One is responsible for the synthesis and assembly of the

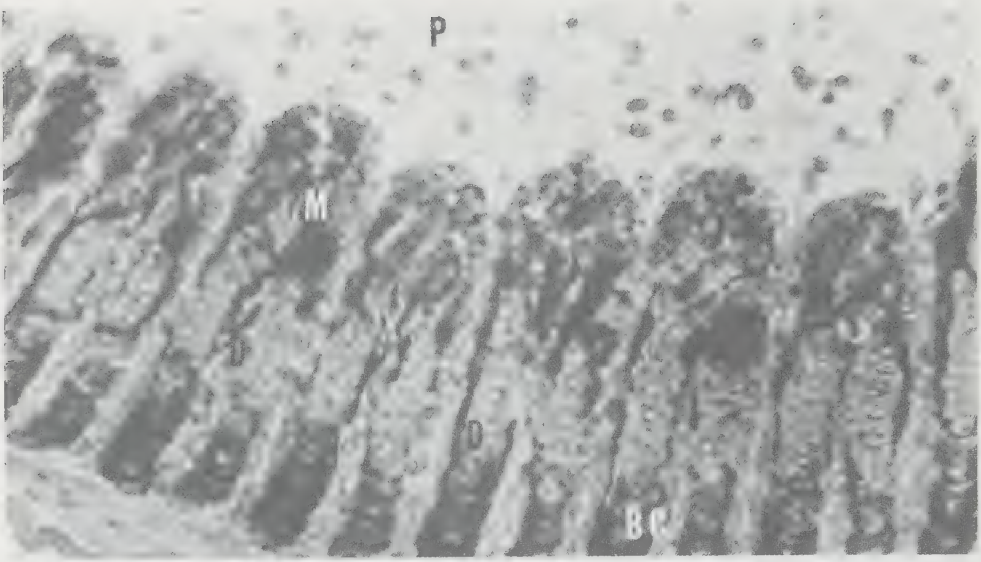

Figure 5.1. Cross section of portion of developing feather showing barb ridges containing melanocytes actively transferring melanin to the developing columns of barbule cells. P - feather pulp; M - melanocyte cell bodies; D - dentrites; BC - barbule cells (570 X).

premelanosomal matrix and the other for the formation and delivery of the tyrosinase-tyrosine complex to the premelanosome (Brumbaugh et al., 1973). Premelanosomes appear to originate in the smooth endoplasmic reticulum. The enzyme tyrosinase is synthesized in the rough endoplasmic reticulum, organized and complexed in the Golgi, and transported by coated vesicles to the melanosomal matrix. When the coated vesicles coalesce with the premelanosomal membrane, the enzyme is activated, and the melanin polymer is synthesized and attached to the matrix (Bowers, 1988).

Melanosome shape and internal structure varies with the melanosomal genotype and the anatomical location of its parent pigment cell. For example, black feather eumelanosomes are rod-shaped, while red or buff feather pheomelanosomes are more spherical (Bohren et al., 1943). Eumelanosomes are also characterized by a highly organized internal structure, while pheomelanosomes have a poorly organized internal matrix, the lighter buff-pigmented granules being the least organized of all (Brumbaugh, 1968). Melanocytes in eumelanized tissues other than feathers produce characteristic melanosomes; for instance choroidal eumelanosomes are larger and more spherical than are those found in feathers (Brumbaugh and Lee, 1975; Boissy, 1986).

Carotenoids

The main carotenoid of the chicken is the yellow fat-soluble pigment called xanthophyll, which has the general formula $C_{40}H_{56}O_2$. Xanthophyll is not synthesized by the fowl, and must be provided by ingestion of such feedstuffs as yellow corn and alfalfa leaf meal. The yellow color of the egg yolk, body fat, skin, shanks and beak is due to xanthophyll (see Lucas and Stettenheim, 1972 for review). Males and nonlaying females store xanthophylls in body fat, skin and blood, but laying females utilize all of their ingested xanthophyll in the production of egg yolks. The relationship between egg laying, and the presence of xanthophylls in the skin and its derivatives, has been used as an indicator of the egg production status of hens.

Carotenoid pigment is also found in several sites in the chicken eye, most noticeably in the iris, where it combines with the vascular system to produce the bay eye color. The photoreceptor cone cells also contain oil droplets containing carotenoid pigment dissolved in a lipid. Droplet color appears to be specific for cone type in the chicken with those in the single cones being red, while yellow and yellowish-green droplets are present in the double cones and their accessory members respectively (see Meyer, 1987 for review). The exact role of these oil droplets in vision has yet to be established.

Vascular Contributions

The heme pigments of blood also contribute to coloration of the eye and the skin and its derivatives. In the eye, both the previously mentioned bay eye and the pink and red eye color of albino and semi-albinos is due to circulating erythrocytes no longer masked by the presence of melanin. Similarly, the red and pinkish colors seen in shanks of some breeds are due to the visibility of dermal vasculature.

Structural Colors

Certain perceived colorations of the fowl are the result of structural variations in the surface of the keratinous feather, rather than due to pigmentation differences. The purple or green iridescent sheens of black feathers are due to the diffraction of light off the feather surface structure (see Lucas and Stettenheim, 1972 for review). That these variations are heritable is shown by the fact that poultry fanciers are able to select successfully for the greenish sheen desired in their show specimens.

GENETICS OF PLUMAGE COLOR

Although both melanins and carotenoid pigments contribute to the feather color of certain avian species, it is the melanins that determine the plumage color and patterns of the domestic fowl. The ultimate presence and distribution of the melanins is complicated by differences in feather form and structure associated with age and sex, as well as structural variations between and within feather tracts and individual feathers. For example, the wild-type phenotype of the red junglefowl is characterized by brown-striped tan down for both sexes, followed by sexually dichromatic feathers resulting in pyle-zoned black-red males and females with a brown-stippled body and a salmon-colored breast. Barred Plymouth Rock chicks are black with a white head spot and do not show their characteristic black and white feather bars until subsequent plumages, while juvenile feathers of pencilled varieties exhibit autosomal barring. Within-feather variation is in part related to structural differences between the webbed pennaceous barbs and the proximal fluffy plumaceous barbs (referred to as undercolor), for instance in the surface white and dark grey undercolor of silver columbian plumage.

A review of our present knowledge of the inheritance of plumage color emphasizes its genetic complexities, particularly the importance of intra- and interallelic interactions. These not only occur between the documented mutations but may also involve genes and gene complexes that are not as yet characterized. It is particularly surprising to find the number of different genetic combinations that can result in a similar phenotype. Furthermore, dominance relationships between alleles and interactions between loci are often only as consistent as their resident genetic background. From the above, it is apparent that the expression of plumage color is a polygenic trait, one in which dominance, epistasis and other gene interactions are important components that culminate in the final phenotype.

Each plumage color and pattern is the result of a series of genetically determined events, and an awareness of these facilitates understanding its inheritance. Based on the wild-type model, genes are presumed to be present for the normal production of melanin and the capacity to produce both eumelanic (black) and pheomelanic (red-buff) pigments. The distribution of eumelanin and pheomelanin appears to be controlled by a genetically determined competitive advantage of one type of pigment over the other. Brumbaugh (1971) has proposed a model whereby eumelanogenesis precedes pheomelanogenesis, thus suggesting that the first genetic decision is the distribution of eumelanin. Pheomelanin pigments the noneumelanic feather tissue, and when it is eliminated by certain mutations (for instance by silver) the result is the striking black and white or greyish plumage patterns. Both types of melanin are subject to dilution effects leading to the blue, buff and cream phenotypes. Totally or partially white plumages also occur as a result of several epistatic mutations, as well as certain genetic interactions.

DISTRIBUTION OF BLACK MELANIN

As suggested by Kimball (1953a), the distribution of eumelanin in the plumage can include either the primary or secondary patterns. The primary pattern refers to the zonal or regional location of black pigment, and may include several feather tracts (as in the columbian restriction pattern) or as few as one (as in flight feathers in the birchen pattern). Secondary patterns are those that affect the distribution of eumelanin within individual feathers, for instance barring, stippling, and lacing. However, it is difficult to classify genes as having only primary or secondary effects, since some genes can affect both types of patterns (Morejohn, 1955).

The *E* locus.

It is convenient to consider the polyallelic *E* locus as determining the basic or zonal distribution of black melanin. The genetics of the *E* locus was misunderstood for years due to the erroneous interpretation of the results of crosses between extended (solid) black and columbian restriction types (where black pigment is restricted to the hackle, wings, feet and tail feathers) (see Hutt, 1949 for review). The epistatic effects of extended black (*E*) were mistakenly considered to be dominance over the columbian pattern with the latter being assigned the allelic symbol *e*. The existence of an *e* allele was first questioned by Cock and Pease (1951), and later eliminated as an *E* allele by the studies of Morejohn (1955), Brumbaugh and Hollander (1965), Smyth and Somes (1965) and Smyth (1970).

It has now been shown that there are at least eight alleles in the *E* series (Table 5.1).

Multiple alleles at the *E* locus were first suggested by Smyth and Bohren (1949) and Kimball (1952a) but both erred by including an *e* allele for the columbian pattern. Morejohn (1955) made a significant breakthrough when he demonstrated the presence of five alleles at the *E* locus, with *E* the most dominant allele ($E>e^+>e^b>e^s>e^y$). The $E>e^b$ association was confirmed by Smyth (1965), while Brumbaugh and Hollander (1965) verified the five Morejohn alleles and added two more, dominant wheaten (e^{Wh}) and buttercup (e^{bc}). The identity of a buttercup allele was further clarified by Smyth et al. (1980), while the eighth allele, birchen (E^R) was described by Moore and Smyth (1972a). Four additional alleles (e^m, e^n, e^{nl} and e^q) have been proposed by Silverudd (1978), but their relationship to the *E* alleles described above, and their separate identity in the absence of associated modifiers, has not been established with certainty.

The adult plumage patterns associated with the different *E* alleles are summarized in

Table 5.1. Adult phenotypes associated with the multiple alleles at the *E* locus.[1]

		Adult plumage	
Name	Gene Symbol	Female	Male
Extended black	E	Solid black	Solid black
Birchen	E^R	Nonblack breaks on head and hackle; body black to finely stippled; marginal nonblack lacing of upper breast of some birchen breeds due to modifying factors	Dark wild-type with dark (black or finely stippled) wing bays (flight feathers)
Dominant wheaten	e^{Wh}	Body varies from light salmon-brown to dark wheat color; a little eumelanin may be present in hackle, wings and tail	Wild-type; same as e^+
Wild-type	e^+	Body feathers have mixture of black and brown pigment in a stippled pattern, while breast is salmon-brown in color and devoid of stippling	Wild type; pyle-zoned black distribution (black breast and ventral plumage); predominantly nonblack hackle, back, saddle, wing bar and wing bay
Brown	e^b	Similar to e^b, but has stippled nonsalmon breast	Wild-type; same as e^+
Speckled	e^s	Resembles e^b pattern, but feathers are less darkly stippled	Wild-type; same as e^+
Buttercup	e^{bc}	Resembles e^b pattern	Wild-type; same as e^+
Recessive wheaten	e^y	Resembles e^{Wh}, although more coarse black stippling present on back and breast	Wild-type; same as e^+

[1] Listed in general order of dominance.

Table 5.1 while six of the eight down patterns are shown in Figure 5.2. Note that only three adult male phenotypes are found, while adult females all differ except for the brown and buttercup patterns. Among the downs, birchen is not shown since it closely resembles the extended black phenotype, while speckled, as described by Morejohn (1955), appears to be intermediate between wild-type and buttercup. In general, dominant wheaten downs show only a faint dorsal head spot, while recessive wheatens frequently display a faint back and head stripe.

The approximate order of dominance is $E > E^R > e^+ > e^b > e^s > e^{bc} > e^y$. The E allele shows nearly complete dominance over all other alleles. Birchen (E^R) is incompletely dominant to e^{Wh} and e^+, the E^R/e^{Wh} genotype in some cases resembling the dark brown (e^b) pattern, but it is dominant over the rest of the alleles. Dominant wheaten also interacts with e^+ and e^b when heterozygous to produce reduced wild-type striped down and an intermediate adult female pattern. Wild-type is completely dominant to e^b, e^s, e^{bc} and e^y, while e^{bc}/e^y heterozygotes are intermediate in phenotype. The degree of dominance appears to be influenced by modifying genes capable of enhancing or inhibiting the expression of eumelanin as suggested by Carefoot (1981).

The existence of two wheaten alleles with similar phenotypic effects, but marked differences in their dominance relationships, is unusual and interesting. Recessive wheaten was first described by Cock and Pease (1951) who extracted it from Light Sussex and called it 'retarded'. It was further characterized by Morejohn (1953, 1955) and Brumbaugh and Hollander (1965). Dominant wheaten was described by Kimball (1952b, 1960b), but it was assigned a separate dominant gene symbol Wh prior to clarification of its E locus relationship by Brumbaugh and Hollander (1965). In contrast, Carefoot (1981) concluded from the results of crosses involving a single Light Sussex bantam that wheaten can act as either a dominant or recessive due to residual modifiers. However, consistent results using both e^{Wh} and e^y by the author indicate that either two separate alleles do exist or that a single wheaten gene is very closely linked to a modifier of eumelanic expression.

Early developmental studies showed that pigment cell differentiation and the type of pigment produced are influenced by both a melanocyte's genotype and its cyto-environment (see Hutt, 1949 for review). Embryonic transplant studies have also shown that the E alleles are autonomous, that is pigment cell synthesis is determined by its genotype and it is unaffected by the host environment (Brumbaugh, 1967; Barsomian, 1983). The sexual dichromatism characteristic of E locus-associated phenotypes is estrogen-dependent, while feather tract differences within a plumage appear to be related to response differences to thyroid hormone, and ultimately to feather growth rate (see Lucas and Stettenheim, 1972 for review). Brumbaugh (1967) demonstrated the role of feather growth rate in the control of black-red differentiation in e^+/e^+ birds, with slow movement through the zone of differentiation favoring pheomelanin synthesis. Extended black (E) was insensitive to the cyto-environment, while recessive wheaten (e^y) mostly differentiated into pheomelanin-producing cells. Another interesting sex difference associated with the E alleles is the two apparently different pheomelanins. One is the typical red pigment of wild-type male plumage, while the other is the brownish-salmon pigment of the e^+/e^+ female breast feathers (Brumbaugh and Moore, 1969) and the wheaten female body feathers (Malone and Smyth, 1979). Smyth et al. (1951) reported previously that wild-type male and female pheomelanins differed in trichochrome content with the salmon-colored feathers containing only small quantities.

When the range in the phenotypic effects of the E alleles are considered, the complexity of this locus becomes apparent. It is difficult to ascertain its functional role in the melanogenic pathway based on the variable expressions of its known alleles. A regulatory

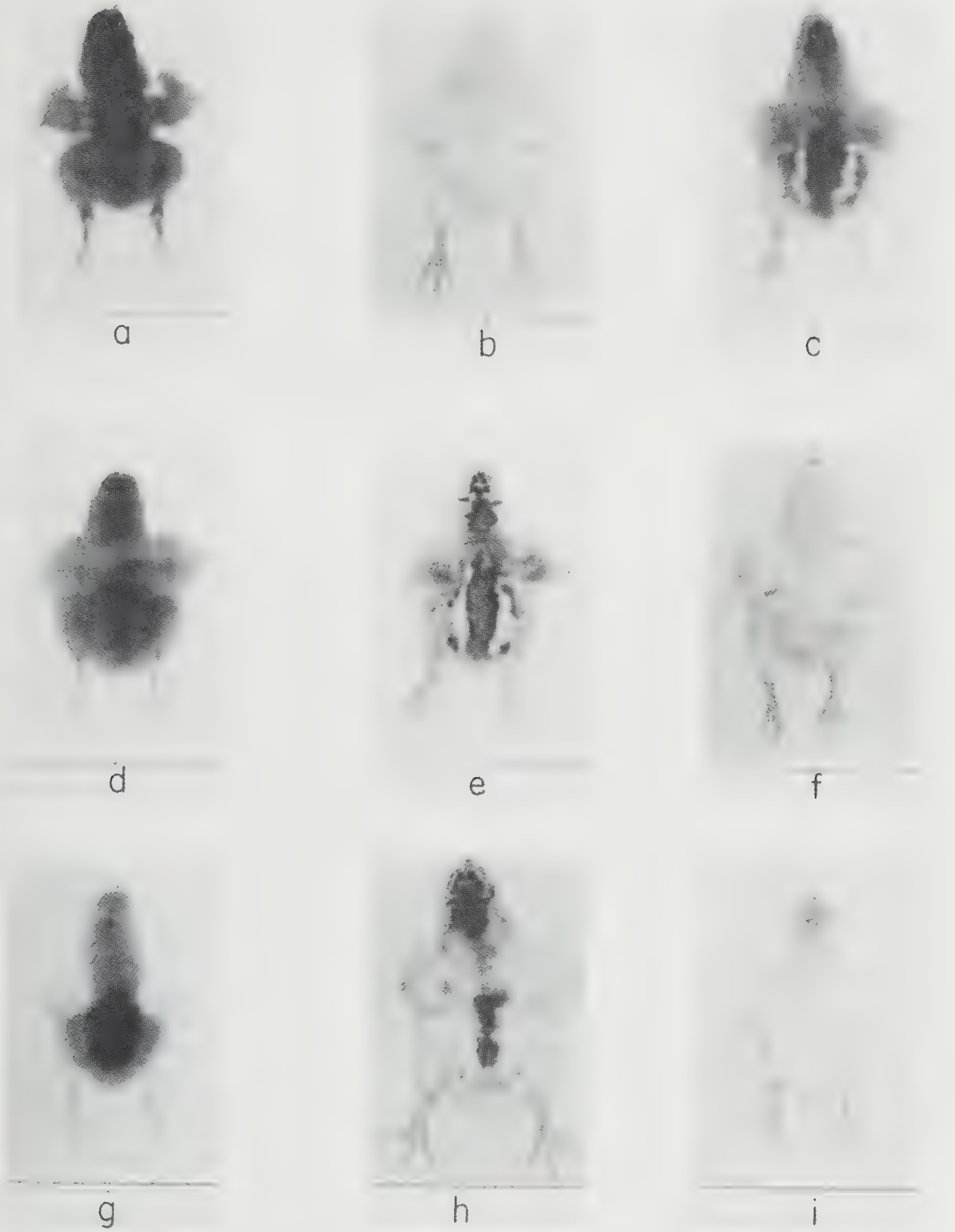

Figure 5.2. Chick down phenotypes associated with the multiple allelic *E* locus, *Co*, *Db* and the *Co-Db* interaction. (a) Extended black, *E*; (b) Dominant wheaten, e^{Wh}; (c) Wild-type, e^{+}; (d) Brown, e^{b}; (e) Buttercup e^{bc}; (f) Recessive wheaten, e^{y}; (g) Columbian, e^{b}/e^{b} *Co/Co*; (h) Dark brown (columbian-like restrictor), e^{b}/e^{b} *Db/Db*; (i) *Co-Db* interaction, e^{b}/e^{b} *Co/Co Db/Db*. (Photographs courtesy S. Lyford).

role for the *E* alleles has been proposed by Brumbaugh et al. (1979) and Wilkins et al. (1982), whereby at the cellular level they control the type or quality of the matrix of the premelanosome. This hypothesis was based on the numerous studies of Dr. John Brumbaugh and his students on melanogenesis in chicken feathers, plus the use of complementation analyses of both heterokaryon and heterozygous cells in culture. It is also tempting to think of the *E* locus as a gene cluster or linked series of functional DNA units, not unlike the major histocompatibility complex (see Crone and Simonsen, 1987 for review). Such a scheme could account for the groupings of the strongly eumelanizing *E* and E^R, the wheatens (e^{Wh} and e^y) and the wild-type variants (e^b, e^s and e^{bc}). Wild-type females exhibit a patterned back and salmon-colored breast, wheaten females have only salmon-colored body feathers, while the brown, speckled and buttercup females lack the salmon coloring, having secondary patterns throughout. Such variation could be accounted for by recombinations within a complex *E* locus. An interesting gene cluster hypothesis for the *E* locus was proposed previously by Kimball (1954a, 1954b), but was flawed by incorporation of the nonexistent columbian allele (*e*) into a multiple allelic series with *E* and e^+.

Other Eumelanizing Factors

In addition to the *E* and E^R alleles, other genetic factors can cause enhanced feather eumelanization, thus playing an important role in certain primary and secondary patterns. Considering the importance of their contributions, it is surprising that the inheritance of these black-intensifying factors is still poorly understood. The earliest interest in eumelanin intensifiers was in relation to the occurrence of black or nearly black stocks that were not characterized by black down color or the dominant and epistatic effects of the *E* mutation. A number of such phenotypes have been described and studied (Punnett, 1923, 1957; Goodale, 1926; Kimball, 1951; and Smyth, 1976), although conclusive evidence for the mode of inheritance is lacking in all cases. Kimball (1951) referred to his stock as pyle black (in contrast to the pyle-zoned black distribution of wild-type) and hypothesized that they represented wild-type (e^+) in the absence of a stippling modifier. Since available data indicate that stippling is a product of the e^+ allele, Kimball's proposal appears to be incorrect (Morejohn, 1955; Moore and Smyth, 1971). Fanciers of Old English Game chickens breed a similar variety to the above, called Brassy Back. According to Jeffrey (1988) who has studied this pattern, Brassy Backs are homozygous for e^+ and an autosomal recessive eumelanin intensifier. Of the above phenotypes that have been described, only one, the Massachusetts Recessive Black line, is totally black and breeds true (Smyth, 1976). This line is characterized by dark brown down (e^b) and white undercolor in the adult males. When crossed to e^+/e^+ and e^b/e^b stocks, its net effect is 'incomplete dominance'. Three and possibly four genes are hypothesized as contributing to the non-*E* self-black plumage.

From the above studies only one eumelanizing gene, melanotic (*Ml*), appears to have been established. It was first described by Moore and Smyth (1971) after extracting it from a randombreeding recessive white population previously described by Jeffrey (1947). *Ml* is an incompletely dominant autosomal mutation that extends eumelanin into the normally red areas of pyle-zoned fowl, while having little effect on chick down color. Heterozygotes on wild-type (e^+) or brown (e^b) backgrounds are distinctly darker, particularly in the hackle and head; however, Ml/ml^+ has little effect on e^{Wh}/e^{Wh} females. Homozygotes approach self-blackness, but the salmon pigment of the wild-type and wheaten females remains evident.

Since its identification, *Ml* has been found to play important roles in a number of primary and secondary patterns, for instance in the quail pattern (Campo and Orozco, 1986), single lacing (Moore and Smyth, 1972b) and double lacing (Carefoot, 1986a, 1986b).

Unsuccessful efforts to determine the genetic basis for certain 'recessive black' lines (Punnett, 1957; Smyth, 1976) suggest the existence of more than one eumelanin intensifying mutation. The melanotic (*Ml*) mutation by itself does not make a wild-type bird black. Furthermore it is not certain that the 'melanotic' genes utilized in the hypotheses of different investigators are in fact the same. Both Carefoot (1986a, 1987a, 1987b, 1987c) and Crawford (1986) have recently studied melanotic; however, Crawford's eumelanizing gene is very closely linked to pea comb (0.3 percent crossovers), while the *Ml* of Carefoot (1987c) is 46 crossover units from the pea comb (*P*) locus. Linkage relationships of the latter's *Ml* with two other markers, *Pg* and *Db*, are in good agreement with those of Moore and Smyth (1971, 1972c). Thus, Crawford's *Ml* probably represents either a second eumelanin enhancing mutation located on the same chromosome, or a translocation of *Ml* to a site closely linked to the *P* locus. The relationship between the two melanotics needs to be clarified.

Genotypic Routes To Self-Black Plumage

Although there are many examples of self-black plumage among the recognized chicken breeds of the world, their similarity is more phenotypic than genotypic. Undoubtedly most are based on extended black (*E*) or birchen (E^R), although totally black plumage can occur in their absence (Smyth, 1976). The presence of eumelanin-enhancing genes in self-blacks carrying *E* and/or E^R was demonstrated by Cote (1976) who analysed one birchen and nine self-black males from a variety of breeds and genetic stocks. All were found to carry eumelanin-intensifying genes, presumably to bolster the epistatic effects of the *E* and E^R alleles. In this study, none of the *E* heterozygotes developed solid black plumage in the absence of eumelanizing modifiers. Melanotic (*Ml*) was a part of all genotypes except for a self-black from a randombreeding population and a Birchen Modern Game. It was linked with the black-lacing gene (as described by Moore and Smyth, 1972b) except in a Mottled Ancona that was homozygous for *Ml* and nonlacing. Other eumelanin intensifiers were also present, even in the birchen, but they were not identified individually. It appears that self-black phenotypes commonly carry eumelanin intensifiers to assist the extension of black pigment by the *E* alleles.

Eumelanin Restriction Factors

There are a number of mutations that act as eumelanin inhibitors, generally modifying the plumage toward the columbian pattern where black pigment is restricted to the hackle, wing, foot and tail feathers. The seemingly meaningless term 'columbian' became associated with this distribution of black pigment following the Columbian Exposition at the Chicago World's Fair in 1893, where Wyandottes with this phenotype were first exhibited. The several genes that are associated with the columbian-like pattern are particularly effective in the restriction of eumelanin from the body feathers of non-*E* females and the black breast of wild-type males. The three best categorized of the eumelanin inhibitors are columbian (*Co*), dark brown (*Db*) and mahogany (*Mh*), while others like (*Di*) and light down (*Li*) have been described only briefly (Table 5.2). These interact with the *E*

Table 5.2. Genes associated with restriction of eumelanin to the hackle, wings, feet and tail plumage (columbian-like restriction).

Gene				
Symbol	Name	Mode of inheritance	Pheomelanic effect[1]	Effect on extended black (*E*)
Co	Columbian	Incompletely dominant	Orange-gold	Little, if any
Mh	Mahogany	Incompletely dominant	Darkens (reddens)	Little, if any
Db	Dark brown[2]	Incompletely dominant	Orange-tan	Reddens black down; variable restriction in adult
Di	Dilute	Incompletely dominant	Marked dilution	Not known

[1] Effects on wild-type reddish or salmon pheomelanins.
[2] Originally named for effects on self-black (*E*) plumage.

alleles, with each other, and probably with unknown genes, to give rise to the various columbian-like primary plumage patterns. While Davenport (1909) and Knox (1927) utilized dihybrid hypotheses to explain the inheritance of the extreme inhibition of eumelanin in buff breeds, the polygenic contribution to columbian restriction patterns was first convincingly demonstrated by the genotypic analysis of a Buff Minorca male by Brumbaugh and Hollander (1966). In addition, two other eumelanin restrictors (*Co* and *Db*) also play major roles in the genotypes of many of the secondary patterns.

Columbian (*Co*). The *Co* mutation was originally found as a hypostatic gene in a recessive white line, and it was then studied in Buff Brahmas by Smyth and Somes (1965). It is probably identical to the ginger mutation (*Gr*) isolated from a Buff Minorca by Brumbaugh and Hollander (1966), but this has not been established by genetic tests. *Co* results in similar adult phenotypes in combination with the wild-type (e^+), brown (e^b) and dominant wheaten (e^{Wh}) alleles at the *E* locus. Homozygous *Co* results in the standard columbian distribution of eumelanin (see American Standard of Perfection, 1983) with e^b/e^b. The $Co/Co\ e^+/e^+$ male phenotype is also columbian-like, while females are similar, but show some limited stippling on their backs. $Co/Co\ e^{Wh}/e^{Wh}$ differs in that eumelanization is reduced in all columbian-restricted areas, particularly in the hackle of both sexes. Heterozygous *Co* is also relatively effective in male plumage with each of the above *E* alleles, but normally stippled areas of the female plumages show intermediate amounts of black markings. In addition to its effects on eumelanin, *Co* also changes the color of pheomelanin to an orange-gold or ginger (Smyth and Somes, 1965; Brumbaugh and Hollander, 1966; Smyth, 1970; Campo and Orozco, 1984). A single *Co* allele will change the salmon coloration of the wild-type female breast or wheaten body feathers to orange-gold. There are no available data indicating that *Co* has any consistent effect by itself on extended black (*E*).

The expression of *Co* in the down is extremely variable and depends on the presence of other genes. In general, it is the result of interactions between *Co*, the *E* alleles, and undefined eumelanin intensifying modifiers. In the absence of the latter, and in the presence of brown (e^b), the down color on the dorsal surfaces is light grey or cinnamon, depending on whether the chick is silver (*S*) or gold (s^+), respectively. The phenotype is similar on an e^+/e^+ background except that the dorsal back stripe is more prevalent and in some cases the outline of the head stripe is also apparent. In the presence of homozygous wheaten, the back surfaces are essentially clear white or gold. The addition of eumelanin intensifiers causes the dorsal surfaces of brown (e^b) downs to vary from dark grey to black (Malone and Smyth, 1975; Figure 5.2g). At the maximum level of eumelanic expression, even the belly is grey, and the only nonblack area is in the extreme foreface area. The most heavily eumelanized chicks are usually females, and these can easily be confused with heterozygous *E* chicks. The eumelanized version of wild-type down typically shows remnants of the back and dorsal head stripe; however, the effect on dominant wheaten down is limited to a slight darkening on the dorsal surface.

Mahogany (*Mh*). The mahogany (*Mh*) gene was first described by Brumbaugh and Hollander (1966), who isolated it from a Buff Minorca. The author later obtained *Mh* through the generosity of Dr. Brumbaugh to make additional observations on its genetics. The effects of *Mh* on primary pattern in females is to restrict generally the amount of eumelanin in the plumage, particularly on the breast, back, and the wing bows and fronts. In the adult male, *Mh* restricts black from the base of the pennaceous part of the breast feathers. The amount of black on the distal ends of the breast feathers shows a dosage effect with the homozygote resembling the spangling secondary pattern. In both sexes, *Mh* causes a general reduction in eumelanin in the back and wing plumage. An additional effect of mahogany is to darken pheomelanic pigment with certain genotypic combinations resulting in a dark red feather color. Down color does not appear to be affected by *Mh* (Brumbaugh and Hollander, 1966).

Dark brown (*Db*). Originally extracted from autosomally barred Fayoumi chickens by Moore and Smyth (1972c), *Db* was first observed to change the black down associated with the birchen (E^R) gene to a dark brown color. The dark brown designation is somewhat misleading because after the mutation was isolated and recombined with wild-type (e^+) and brown (e^b), it was found to have a major primary pattern effect, resulting in modified columbian-like eumelanin distribution (Moore et al., 1978). Homozygous *Db* adult males have a black-tailed red or silver (s^+ vs. S) phenotype characterized by nonblack hackles and reduced eumelanin in the wings and tail. The homozygous *Db* female pattern resembles a modified columbian distribution of black pigment, differing in the presence of an autosomal barring-like secondary pattern on the ventral surfaces of e^b/e^b breast and back plumage (Moore et al., 1978; Carefoot, 1985). The effects are similar on e^+/e^+ females except that *Db* has relatively little effect on the salmon-colored breast feathers or the salmon-colored body plumage of dominant wheaten females (Moore et al., 1978). Heterozygous *Db* females resemble homozygotes except that the secondary markings are more pronounced. The Db/db^+ males are quite variable in phenotype, most commonly affecting e^+, e^b or e^{Wh} backgrounds by adding a red tip to the black breast feathers. However, variations with more red-brown color do occur, with some birds showing completely red-brown breast plumage (Moore and Smyth, 1972c). Similar variations in the expression of *Db* in males were noted by Campo and Alvarez (1988), but the genetic basis for this remains unclear.

The *Db* mutation also modifies the color of pheomelanin with the resulting plumage exhibiting a distinctive orange-tan or burnt-orange appearance (Moore et al., 1978). This is not apparent in the wild-type salmon breast or wheaten body feathers, where the effect is to lighten the salmon coloration only. Homozygous wild-type, brown, and dominant wheaten males all show a slight but consistent fading in the back region due to an absence or marked dilution of pheomelanin on the distal margins of the saddle feathers.

All of the downs associated with the *E* alleles are modified by *Db*, particularly when homozygous (Moore and Smyth, 1972c; Moore et al., 1978). The effect on extended black (*E*) and birchen (E^R) is to modify the black down to a dark brown. This may vary from a dark brown foreface to a completely brown chick, depending on *Db* dosage and the residual genotype. Although *Db* typically acts as a dominant, Campo and Alvarez (1988) reported that in the presence of E^R it acted as a sex-influenced gene, being dominant in males and recessive in females. In the presence of wild-type or brown, *Db/Db* results in markedly reduced brown dorsal back stripes on a light cream background (Figure 5.2h). Heterozygotes are indicated by widened yellowish white back stripes, and in brown (e^b) downs, lightened tan areas in the foreface and adjacent to the forecomb. In the presence of homozygous buttercup (e^{bc}), *Db* leaves only a few head spots and a faint lateral back stripe on a cream background (Smyth et al., 1980). *Db* is not expressed in wheaten down.

Dilute (Di). Brumbaugh and Hollander (1966) extracted another incompletely dominant mutation, dilute (*Di*), during their genetic analysis of a Buff Minorca. Although the information concerning *Di* is limited, it appears to be a partial restrictor of eumelanin as mature dilutes were described as being reddish. This is also suggested by the fact that *Di* removes dermal melanin from the shanks, thereby mimicking the inhibitor of dermal melanin (*Id*) mutation. Wild-type chicks carrying *Di* had pale down that lacked the lateral black back stripes, although a fairly well defined head and eye stripe remained.

At the present time, the relationship of *Di* to other mutations or gene interactions must await additional research. Interestingly, Carefoot (1985) described males of a pullet-producing mating for show quality Pencilled Wyandotte bantams as having diluted pheomelanin and down color similar to the description for dilute provided by Brumbaugh and Hollander (1966).

Other columbian-like restrictors. There are undoubtedly other genes that tend to restrict eumelanin from the breast and/or body plumage. Surprisingly, some of these appear to be carried as hypostatic genes under extended black-associated plumage. In a genetic analysis of nine homozygous or heterozygous *E* males each representing a different breed, variety or gene pool line, Cote (1976) found eight to carry columbian-like modifiers, particularly effective for breast feathers. These cases showed considerable variation in their penetrance and expressivity, although all of them restricted eumelanin in wild-type (e^b) segregates from F_1 and backcross test matings. Columbian restriction phenotypes among the eight progeny groups could be grouped into one of three general types: those that were expressed only in e^+/e^+ chicks and adults, E/e^+ progeny being typically black; those that showed restriction of adult E/e^+ progeny, but no effects on black down; and those that affected both E/e^+ down (brown faces or heads) and adult plumage. Although the parent males differed in their eumelanizing modifiers, these data strongly suggest the presence of a number of different columbian-like restrictors. Test matings to dark brown (*Db/Db*) and columbian (*Co/Co*) tester lines indicated that none of the males carried *Db*, while only two carried *Co*. One of the latter was an exhibition-bred Barred Plymouth Rock. This is interesting in light of the observation of Smyth and Somes (1965) that *Co* enhanced the expression of sex-linked barring (*B*) on an e^+/e^b background.

Genetic Basis For Some Columbian Restriction Phenotypes

Although there is a range in the amount of eumelanin present within the broad category of columbian-like restriction, three general groupings have been suggested by Smyth (1970). These include: 'standard columbian' breeds with heavily eumelanized hackles, wings and tails, as in Buff and Light Brahmas, and Columbian Plymouth Rocks; the 'black-tailed reds' with relatively reduced hackle eumelanization, as in New Hampshire, Rhode Island Red, Red Sussex, and Spanish Vasca; and the 'buffs' with essentially an absence of eumelanin in all columbian-restricted areas, as in Buff Orpington, Buff Plymouth Rock, and Buff Minorca.

Although it is now possible to identify key primary pattern genes that explain genetic differences among the above groups, as well as breed differences within them, unidentified modifiers still contribute to pattern variations seen within the breeds and varieties. Following an extensive series of crosses between three columbian-like breeds differing in the amount of eumelanin in the hackle, wings and tail, Somes and Smyth (1966) proposed that these phenotypes were controlled by polygenic differences. The three breeds included Rhode Island Red (with the most black), New Hampshire (with an intermediate amount of black) and Buff Orpington (with no black). These workers were not aware at that time of the roles played by specific columbian restriction genes. They did propose the existence of an innate antero-posteriad threshold that allowed a step-wise increase in eumelanin distribution from hackle to tail, with the anterior portion having the highest threshold. Inherited variations in eumelanin depositing potential were then thought to interact with the threshold to produce the varying amounts of eumelanin in the appropriate feathers. It is likely that such a mechanism still functions within the guidelines determined by such primary pattern genes as *Co*, *Mh* and *Db*. Also of interest in this study was that all three breeds carried a common columbian restriction genotype; all adult patterns observed in the F_2, and two following generations of randombreeding from pooled F_2 offspring, were columbian-like in phenotype. This was not the case for down color where a few phenotypes showing more eumelanin than the original parent lines were observed (Somes, 1969).

Established and hypothesized genotypes for some specific columbian restriction phenotypes follow:

Buff or Light Brahma - $e^b/e^b\ Co/Co\ db^+/db^+\ mh^+/mh^+$. These represent typical standard-type columbians that were shown to be $e^b/e^b\ Co/Co$ by Smyth and Somes (1965) and verified by Moore and Smyth (1972b). The absence of *Db* and *Mh* is based on subsequent observations by the author.

New Hampshire - $e^{Wh}/e^{Wh}\ Co/Co\ db^+/db^+\ Mh/?$. A black-tailed red reported to be e^{Wh} and *Co* by Smyth (1970). Since that time, *Mh* has been found in the same line by the author.

Rhode Island Red - $e^y/e^y\ Co/?\ db^+/db^+\ Mh/Mh$. This breed resembles a darker version of the New Hampshire and was found to be recessive wheaten (e^y) by Brumbaugh and Hollander (1966). Homozygosity for *Mh* and segregation for *Co* was demonstrated by Malone (1975) in an exhibition-bred stock.

Prat - $e^{Wh}/e^{Wh}\ Co/Co\ db^+/db^+\ mh^+/mh^+$. This Spanish breed is characterized by orange-buff down and ginger-red adult plumage. Its homozygosity for e^{Wh}, *Co* and db^+ was determined by Campo and Orozco (1984).

Vasca - $e^{Wh}/e^{Wh}\ Co/Co\ db^+/db^+\ Mh/?$. The Vasca, like the Prat, is reported to carry e^{Wh} and *Co*, but adults are darker, resembling the New Hampshire (Campo and Orozco, 1983). Pheomelanin color differences appear to be due to a single gene that is dominant for darker coloration. These workers suggest that this could be due to the presence of different *Co* alleles, or to some other major modifier (possibly Mahogany (*Mh*), author's suggestion).

Villafranquina - $e^b/e^b\ co^+/co^+\ Db/Db\ mh^+/?$. Another Spanish black-tailed red characterized by a large amount of secondary pattern on female back plumage (due to *Db*) and analyzed by Campo and Alvarez (1988).

Buff Minorca - e^y *or* $e^{bc}/?$ Co/Co db^+/db^+ Mh/Mh Di/Di. A noneumelanic version of columbian restriction was analyzed by Brumbaugh and Hollander (1966). The particular bird studied was heterozygous for buttercup (e^{bc}) and recessive wheaten (e^y) at the *E* locus. *Co* has been tentatively substituted for their proposed *Gr* symbol (ginger). There is no evidence that *Db* was present in the bird analyzed.

Additional eumelanotic columbian types. Preliminary evidence indicates that several stocks are heavily eumelanized versions of columbian restriction. A melanotic version of the Prat breed (e^{Wh}/e^{Wh} Co/Co) was found to be homozygous for melanotic (*Ml*) by Campo and Orozco (1986). These workers suggest a similar genotype for the quail pattern of the Belgian Quail bantam breed. In contrast, Kimball (1954a) concluded that the quail pattern represented a combination of birchen (E^R) and columbian ('*e*'). The phenotype of the Lakenvelder, a heavily eumelanized columbian breed carrying silver (*S*) instead of gold (s^+), is suggested to be e^b/e^b Co/Co plus unidentified eumelanizers (Smyth, unpublished). The Lakenvelder phenotype regularly appeared among progeny of a gene pool stock that was homozygous for brown (e^b), but segregating for columbian (*Co*) and melanotic (*Ml*) and additional eumelanin enhancers capable in the absence of *Co* of producing the recessive black phenotype.

Further evidence for the flexibility of genotypic determination of phenotype has been furnished by studies of columbian restriction patterns. The finding that a Buff Minorca was heterozygous for e^{bc} and e^y (Brumbaugh and Hollander, 1966), suggests that primary columbian pattern genes plus modifiers are capable of masking more than a single type of *E* allele. Also of interest is an exhibition Rhode Island Red analyzed by Malone (1975) that was heterozygous for dominant (e^{Wh}) and recessive wheaten (e^y), as well as being Co/co^+.

Secondary Patterns

Secondary feather patterns are those that determine the distribution of eumelanin within individual feathers. A number of these are depicted in Figure 5.3. Many of these exist alternatively in the silver or gold version depending on whether pheomelanin is present or absent in the nonblack feather tissue. At the developmental level, all of these represent some type of barring activity occurring in the long axis of the feather during its barb formation (Kimball, 1953a). The genetic processes that turn eumelanization on and off vary from the multiple black and white bars of the Barred Plymouth Rock feather barbs to the single switch turning off eumelanin production in the barbs of single-laced varieties such as the Laced Wyandotte. Chickens featuring secondary patterns have long been a favorite of poultry fanciers, for their aesthetic beauty and the challenge of breeding the complicated and precise patterns set forth by breed standards committees. This has led to the common practice of keeping different genetic sublines within a specific breed or variety for use in separate matings to produce specific male and female plumage patterns as set forth in the various standards of perfection.

Most secondary plumage patterns of chickens are determined by autosomal inheritance. Their striking phenotypes were subjected to genetic analyses by a number of pioneering geneticists, including Professor R.C. Punnett, whose book *Heredity In Poultry* (1923) should be required reading for all aspiring to work in this field. These earlier studies were thoroughly reviewed by Hutt in 1949, and the resulting conclusions provided a sound base on which to build. A role for interactions between secondary pattern genes at different loci was suggested by Kimball (1953a, 1959) to explain the complexities associated with the

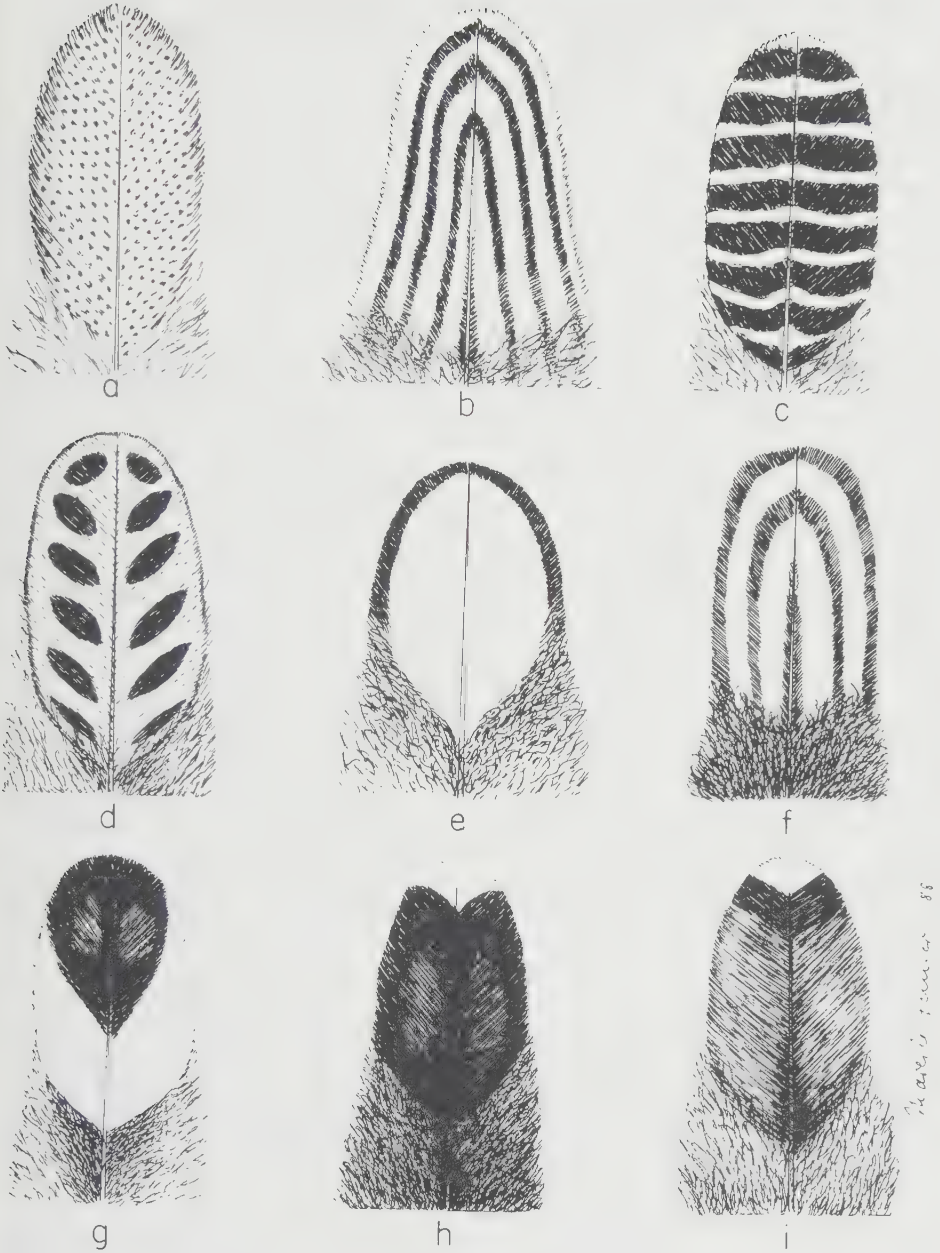

Figure 5.3. Secondary pattern genes: (a) stippling, (b) pencilling (concentric), (c) autosomal barring - also called parallel pencilling, (d) buttercup - autosomal barring modification, (d) single lacing, (f) double lacing, (g) spangling, (h) mottling, (i) tricolor pattern as in Mille Fleur and Speckled Sussex.

inheritance of stippling, pencilling and autosomal barring (Figure 5.3a, 5.3b, 5.3c). Later, Moore and Smyth (1972b) showed that the single lacing pattern of Silver Laced Wyandottes could be explained by the combined contributions of mutations from four loci. Somes (1980) demonstrated that a single mutation, mottling (*mo*), could interact to result in five different phenotypes. Much of our recent progress has resulted from the identification of the alleles at the *E* locus, key eumelanin restrictor mutations (*Co, Db* and *Mh*), and the understanding of some of the interactions between these factors. Building on this, Dr. W.C. Carefoot, an English breeder, exhibitor and judge of fancy poultry, has engaged in a number of studies on the inheritance of secondary patterns. The result is that Carefoot, a private businessman with a doctorate in mathematics, has come forth with an interesting and innovative series of genetic hypotheses (Carefoot, 1985, 1986a, 1986b, 1987a, 1987b, 1987c, 1988).

In summarizing plumage color inheritance in his 1949 book, Hutt followed the trend of the times and listed individual genes for lacing (*la*), autosomal barring (*ab*), mottling (*mo*), pied (*pi*) and spangling (*Sp*). Stippling was considered to be wild-type and no explanation for the inheritance of crescentic pencilling was offered. Kimball (1953a, 1953b, 1956c, 1959) pointed out that a number of the secondary patterns genes showed incomplete dominance, and proposed the symbols *Lg* and *Bg* for lacing and autosomal barring, respectively, while adding stippling (Sg^+) and pencilling (*Pg*) loci. However, all of his secondary pattern studies were flawed by the use of e^+/e^+ (pyle-zoned black) at the *E* locus, and his failure to understand the columbian restriction genes *Co* and *Db*. Moore and Smyth (1972b, 1972c) utilized different *E* alleles, along with *Co*, *Db* and *Ml* (melanotic), while using the previously proposed pattern genes *Lg* and *Ab* to explain single-laced and autosomal-barred patterns. Carefoot (1985) studied crosses between Partridge Wyandottes, a pencilled breed, and Gold-Pencilled Hamburgs, a misnamed autosomal-barred breed, and found them to differ only in that the latter carried the columbian-like gene *Db*. In other words, they carried the same pattern gene to which Carefoot assigned the symbol *Pg*, and they differed by the *Db-Pg* interaction found in the barred breed. The stippling pattern of the wild-type, therefore, resulted from the absence of the *Pg* pattern gene, being genotypically pg^+/pg^+. He then showed that lacing was also due to *Pg* with the single and double lace forms (Figure 5.3e, 5.3f) resulting from the presence or absence of *Co*, respectively (Carefoot, 1986a, 1986b, 1987b). In concert with the eumelanin enhancer melanotic (*Ml*), *Pg* was shown to be a part of the lacing genotype by Moore and Smyth (1972b). Carefoot (in press) has further proposed that *Pg* is the secondary pattern gene for spangling (Figure 5.3g).

The inheritance of secondary plumage color genes is presented in more detail below and also related to representative phenotypes in Table 5.3. Although the major individual genes associated with specific patterns have been demonstrated, it should be noted that unidentified modifying genes still are needed to produce high quality phenotypes. *Pg* has been shown to be in linkage group III on chromosome 1 and is linked with the columbian-like restriction gene *Db*, and with melanotic (*Ml*) by approximately 20 and 10 crossover units respectively, with the *Ml* locus in the middle (Carefoot, 1987c).

Sex-linked barring. Sex-linked barring is not only one of the most attractive of the secondary patterns, but the most commercially useful because of its value in matings designed for sex identification in the chick down. It is a breed characteristic of the Barred Plymouth Rock, Dominique, Delaware, Old English Crele Game, Scots Gray, and several cuckoo-patterned varieties.

The sex-linked form of barring was established as a dominant gene in 1908 by

Table 5.3. Genetic interactions that determine the autosomal secondary patterns in the domestic fowl.

	Genotypes[1]					
Phenotype	E alleles[2]	Pg/pg^{+}[3]	Co/co^{+}[4]	Db/db^{+}[4]	Ml/ml^{+}[5]	Mo^{+}/mo[6]
Stippling	e^{+}	pg^{+}	co^{+}	db^{+}	ml^{+}	Mo^{+}
Pencilling	e^{b}	Pg				
Autosomal barring:						
Hamburg type	e^{b}	Pg		Db		
Fayoumi type	E^{R}	Pg	Co	Db		
Buttercup	e^{bc}	Pg		Db		
Single lace	e^{b}	Pg	Co		Ml	
Double lace	e^{b}, e^{Wh}, e^{y}	Pg			Ml	
Spangling	E or E^{R} ?	Pg		Db	Ml	
Mottling	E	?	?		?	mo
Speckling (Sussex)	e^{b} or e^{Wh} ?	?	Co		?	mo

[1] Pure stocks assumed to be homozygous.
[2] See descriptions in Table 5.1.
[3] pg^{+} is wild-type stippling, while Pg allows nonstippled secondary patterns.
[4] Co and Db are the columbian and dark brown 'columbian-like restrictors', respectively.
[5] Ml is the eumelanin enhancer melanotic.
[6] mo is the mottling mutation.

Spillman, and was the subject of many early studies that also showed it to differ from a second, or autosomal type, of barring (see Hutt, 1949 for review). A closely related mutation, called sex-linked dilution was described by Munro (1946), who indicated it to be either an allele of sex-linked barring (B), or very closely linked to it. Van Albada and Kuit (1960) later isolated what appeared to be the same gene, and their data suggested it to be allelic and dominant to B. They suggested changing the locus designation to *Sd*, but the long use of the B symbol, and its recent use in the linkage map of the fowl by Somes (1984), warrants retention of B locus. Therefore, the allelic dominance order is $B^{Sd}>B>b$. The B^{Sd} mutation was referred to as a type of true-breeding blue by Munro, since hemizygous females have light blue and barred plumage as do the heterozygous males; however, homozygous males reflect a dosage effect and are essentially white (Munro, 1946; Van Albada and Kuit, 1960). The latter resemble dominant whites but differ in that they are epistatic to pheomelanin, while dominant white is not.

Barring (B) also shows a distinct dosage effect, indicating a lack of dosage compensation at this locus on the Z chromosome, as pointed out by Cock (1953). Most of the observations and studies on barring have been made on a black (E/E) background as in the Barred Plymouth Rock (BPR). Unless otherwise noted, this will be the case for the following discussion. In adult plumage, B/B males have wider white bars than do B/b^{+} males and B/W females, the latter two having narrower white bars of similar width. Similarly, two B alleles more effectively remove the eumelanin from the scales on the shanks than does one. The parallelism and width of the bars, as well as the sharpness of demarcation along the black and white bar interface, more closely resembles the show standards in slower growing feathers. Therefore, breeders of exhibition BPR have incorporated the sex-linked slow feathering gene K, as well as other modifiers favoring slow feather growth, into their stocks. In the down, black chicks show distinct white dorsal head spots with those of males being larger with less well defined edges, than those of females (see Hutt, 1949 for review). At hatching, female chicks have dark eumelanized shanks with a sharp demarcation between the black and the yellow skin colors on the toes. In contrast, male chicks have shanks that are lighter throughout, and lack the sharp black-yellow toe demarcation of their sisters. Because of the head spot and shank color differences, pure BPR can be sexed at hatching with accuracy as high as 98 percent (Jerome, 1939). Because of its dosage effect, *B* has been introduced onto other genetic backgrounds to develop the so-called autosexing breeds, that also can be sexed at hatching without the need of crossbreeding (for review see Hutt, 1949; Cock, 1953; Silverudd, 1978).

The *B* gene is autonomous, since wing bud transplants from BPR embryos to nonbarred embryos remain barred and even express their appropriate dosage effect (Danforth and Foster, 1929; Willier, 1941). Nickerson (1944) suggested that the rhythmic control of eumelanin deposition in the barred feather was due to some diffusible factor from the pigment cell that would build to a critical level and inhibit normal melanization. The adjacent white bar would be the result of feather growth during the period necessary to clear the inhibiting factor. Following a series of studies, Bowers (see Bowers, 1988 for review) agrees that a cytotoxic effect is involved, but believes that *B* melanocytes are simply more sensitive to the build-up of normal levels of melanin precursor products, which are known to be toxic to pigment cells (Hochstein and Cohen, 1963; Lerner, 1971). The distal end of all barred feathers starts with a black band and the melanocytes adjacent to the interface with the white band have a high tyrosinase activity, as well as a high phosphatase activity, suggestive of autolysis, when compared with pigment cells in the rest of the black band (Bowers and Asano, 1984). Since the white band is devoid of melanocytes (Novoa and Bowers, 1988), sensitive *B* cells would be prevented from migrating into this area until the precursor build-up was cleared. Nonbarred (b^+) melanocytes are presumably insensitive to the build-up of normal eumelanin precursors. In support of Bower's contention, BPR melanocytes have been shown to survive normally in culture when removed from their *in vivo* cytoenvironment (Bowers and Gatlin, 1985). The sex-linked *B* allele also reduces melanin levels in the eye in a dose dependent manner (Stradtner et al., 1988). According to these workers, *B*-induced choroidal hypomelanosis is the result of two different mechanisms: a reduced number of pigment cells due to an inhibition of melanoblast migration from the neural crest, and decreased melanogenic activity after populating the choroid.

The expression of *B* on non-*E* backgrounds is more variable, but in general, black feathers are barred in the presence of *B*, but the expression of the barring is less precise, as in the Old English Crele Game. Barred red or buff columbian stocks have been developed, including the Gold Barred Rock (Punnett and Pease, 1928) and the Gold Barred Basque (Campo and Orozco, 1980). The expression of head spotting is more variable on pheomelanic backgrounds, and is enhanced in chicks with darker head coloration (Punnett and Pease, 1928; Jaap, 1941). Barring also is expressed, although again imprecisely, on autosomal blue (Bl/bl^+) feathers (Lippincott, 1921; Smyth, unpublished). This probably explains the lack of popularity of blue barred breeds among breeders of exhibition poultry.

Stippling. The red junglefowl is characterized by sexually dichromatic plumage. Males are pyle-zoned black with pheomelanin present in the areas of sexually dimorphic feather structure. Females are finely stippled with dark and light brown (Figure 5.3a) except that the breast feathers are salmon-colored and lack the stippling pattern. Wild-type chick down shows prominent dorsal, lateral back, and head stripes of dark brown pigment on a lighter tan ground color. These phenotypes are inherited together under the designation of e^+. In the absence of major modifiers like *Co* or *Db*, a wild-type chick will develop into a stippled, salmon-breasted female or a pyle-zoned wild-type male. The association of striped down with the female salmon breast suggests the activity of single allele; however, stippling can occur on other genetic backgrounds, for instance brown (e^b), which differs from wild-type in down color and by the absence of the salmon breast. Kimball (1953a, 1959) believed that an incompletely dominant gene Sg^+ was responsible for the stippling pattern, while its recessive allele *sg* eliminated stippling from wild-type female feathers without altering the primary pattern distribution of eumelanin. He reported working extensively with *sg*, and that e^+/e^+ *sg*/*sg* (pyle-blacks) were nonstriped black in the down, a phenotype that the author has never encountered. In contrast, Carefoot's (1985) proposal of a single mutation, *Pg*, that alters the stippling process to allow a number of genetically modified rearrangements of eumelanin within the feather, appears more plausible.

Whether or not the development of stippling in a feather can be attributed to a single gene, cannot be ascertained at this point. Based on present knowledge, however, stippling can be designated as pg^+, with no known mutant allele for absence of secondary pattern.

Pencilling and autosomal barring. The terminology associated with the pencilling and autosomal barring patterns has been confused by the exhibition poultry breeders' referral to both phenotypes as pencilling. One is characterized by concentric rings wholly contained within the feather (Figure 5.3b), while the other is the parallel type (Figure 5.3c) referred to by geneticists as autosomal barring. Therefore, Silver Pencilled and Partridge Plymouth Rock females have concentric-pencilled feathers, while their male counterparts are wild-type. In contrast, Silver Pencilled Hamburg females are autosomal barred and the males are columbian-like with black restricted to the wings and tail. Interestingly, pencilled and autosomal barred birds both display autosomal barring in their juvenile plumage. Pencilling and barring, as referred to herein, will indicate the concentric and parallel types, respectively.

Kimball (1953a) suggested that an incompletely dominant gene *Pg* reorganizes stippling into the pencilled pattern in females. Carefoot (1985) concurred, but attributed a wider secondary pattern role for *Pg*. The pencilled breeds that have been tested have been e^b/e^b (brown) at the *E* locus. Although the author is not aware of pencilled e^+/e^+ varieties, one would expect e^+/e^+ *Pg*/*Pg* adult females to have pencilled body feathers except for those in the salmon breast area.

Carefoot (1985) concluded that barring differed from pencilling by the addition of the *Db*/*Db* columbian-like restrictor to the non-stippled pattern genotype (*Pg*/*Pg*). Moore and Smyth (1972c) had isolated *Db* from Fayoumi fowl, an autosomal barred breed, and reported it to be linked to a generally accepted autosomal barred gene *Ab* (see Hutt, 1949 for review) by 17 crossover units. In contrast, Carefoot (1985) considered *Db* to be the inducer of the barring pattern and later showed a similar linkage relationship between *Db* and his *Pg* (Carefoot, 1987c). In a later study, Smyth et al. (1980) showed *Db* to be associated with the modified autosomal barred pattern of the Buttercup breed (Figure 5.3d). It was also found in an autosomal barred Gold Pencilled Hamburg male (Smyth, unpublished). The Fayoumis were E^R/E^R at the *E* locus, while the Buttercups carried the buttercup allele, e^{bc}. The Gold Pencilled Hamburg proved to be heterozygous at the *E* locus (e^b/e^{bc}), although Carefoot (1985) determined another strain of Gold Pencilled Hamburgs to be homozygous for e^b. The genetic basis for the difference between the parallel bars of the Hamburg and the modified ovate spotting of the Buttercup is not known, but it is presumed to involve other modifying genes. Jaap and Hollander (1954) had previously proposed that buttercup and autosomal barring differed by presence of an 'autosomal black' gene, which presumably would lead to the greater expression of the parallel bars in barred feathers. Kimball (1953b, 1959, 1960a) also studied the buttercup pattern and proposed a complex allelic relationship between stippling and pencilling genes at a single compound locus and an autosomal barring gene located at a different locus. He thus explained a number of stippled, pencilled and barred patterns; however, no supportive genetic data were presented.

The difficulties in classifying segregating phenotypes from matings involving stippling, pencilling, and autosomal barring is probably related to the residual genetic variation. Carefoot has used relatively homogeneous exhibition-bred stocks for his studies, thus undoubtedly simplifying progeny classification. The F_2 offspring from a mating between a synthetic brown ($e^b/e^b\ pg^+/pg^+$) male and Partridge Plymouth Rock females, from a line characterized by considerable variation in the quality of pencilling, resulted in F_2 females that in many cases were difficult to classify (Smyth, 1965). Variable results among the F_2 progeny of a cross between Silver Sebright (single laced) and Gold Pencilled Hamburgs (autosomal barred) were also reported by Punnett (1923). He also noted that variable pencilling was found in some birds, especially in feathers at the base of the tail.

Lacing. Two general forms of lacing, single and double (Figure 5.3e, 5.3f), are recognized by poultry fanciers. In the single form an outer ring of eumelanin conforms to the edge of the feather, the nonblack area being white or gold depending on the genotype at the *S* locus. The single lace pattern appears in both sexes but is modified in the sexually dimorphic regions of male plumage so that only the breast and ventral feathers of males are typically laced. The presence of henny feathering (*Hf*/*Hf*), as displayed by the laced Sebright bantams, results in complete lacing in both sexes. Double-laced feathers add an inside lace separated by a noneumelanic space between it and the outer lace. A remnant of a third lace follows the rachis in certain feathers. The double-laced phenotype is present in the female only, males being typically wild-type.

Kimball (1955) reported lacing to be due to an incompletely dominant gene (*Lg*), although Hutt (1949) had earlier considered it to be due to a recessive gene, primarily on the basis of the results of a cross reviewed by Punnett (1923). Among 299 F_2 progeny from a cross between Silver Sebright and Gold Pencilled Hamburg, Punnett recovered only 44 birds with varying degrees of lacing. Only four of these were described as having lacing of parental quality. Actually this is not surprising in light of the demonstration of the complex genetic basis for single lacing in Silver Laced Wyandottes by Moore and Smyth (1972b). The latter found that homozygosity for four genes, brown (e^b), columbian (*Co*), melanotic (*Ml*) and lacing (*Lg*), were necessary for single lacing in the Wyandotte and that the *Ml* and *Lg* loci were linked by approximately ten crossover units.

Previously, observations on a gene pool population segregating for a number of color mutations had indicated that single lacing occurred only in feathers from which eumelanin was restricted by the *Co* gene (Smyth, unpublished). Moore and Smyth (1972b) verified that *Co* was necessary for expression of single lacing. They also found that by itself the lacing gene caused an incomplete lace which was restricted to the distal tip of the feather. Melanotic (*Ml*) was required to complete the lace, with the two genes functioning dose-dependently.

Carefoot (1986a, 1986b) confirmed the findings of Moore and Smyth (1972b), but reported that their *Lg* gene was actually his previously reported secondary pattern gene *Pg*. Therefore, he contended that *Co* is the instigator of single lacing through its interaction with *Pg*. Carefoot (1987c) also showed that the *Pg* and *Ml* loci had the same linkage relationship as that reported for *Lg* and *Ml* by Moore and Smyth (1972b).

Double lacing was believed by Kimball (1955) to result from his version of lacing (*Lg*) and pencilling (*Pg*) on an e^+/e^+ background. *Pg* was proposed to add the inner lace. However, Carefoot (1986a) showed that removal of *Co* from the e^b/e^b *Co*/*Co* (*Ml-Pg*/*Ml-Pg*) genotype for single lacing resulted in double-laced females and wild-type males. The particular effectiveness of *Co* as an eumelanin restrictor in the male wild-type pattern had been reported previously (Smyth and Somes, 1965; Smyth, 1970).

Although the laced patterns in the Wyandotte and Barnevelder breeds have been shown to be on an e^b/e^b background (Moore and Smyth, 1972b; Carefoot, 1986a, 1987b), double lacing can also be expressed on a wheaten background. Double lacing is the secondary pattern of the Dark Cornish variety, reported to be recessive wheaten (e^y) by Morejohn (1955), although another strain appeared to be homozygous for dominant wheaten (e^{Wh}) when testmated to e^+/e^+ (Smyth, unpublished). Carefoot (1988) has also shown that the black single lacing characteristic of the Blue Andalusian is due to *Co* and the *Ml-Pg* combination. This breed is *E*/*E* with heterozygosity for the blue mutation (*Bl*) responsible for the modification of eumelanin to the blue-grey phenotype.

Spangling. The spangling pattern consists of a V-shaped eumelanic spangle located at the distal end of the feather (Figure 5.3g). The remainder of the webbing, as was the case for lacing, may be white, or some shade of pheomelanin. This attractive pattern is characteristic of the Silver Spangled and Golden Spangled Hamburg varieties. The two varieties also differ in that the rectrices, sickles, and tail coverts are solid black in the golden variety, but white with a black-spangled tip in the silver variety. Although the reason for this relationship is not known, it must be very basic (either developmental or very close linkage) as Taylor (1932a) never observed a definite spangled tail in any of his gold segregates. Females of both varieties have a larger spangle than do males, an apparent consequence of estrogen-enhanced eumelanic activity rather than a sex-linked effect (Taylor, 1932a).

The down of spangled chicks is characterized by irregular or 'broken' back striping. The head down of the silver variety is essentially clear, while the golden chicks show varying degrees of a dorsal head streak. Spangling is not expressed in the early chick feathers, being first apparent at approximately four weeks of age (Dunn and Landauer, 1930).

Early studies on the inheritance of spangling are somewhat confusing, particularly in regard to the *E* allele present; however, Punnett (1923) and Taylor (1932a) both concluded that the pattern was due to an autosomal dominant gene, *Sp*. Taylor considered spangled birds to have a columbian-type restriction, but his data indicate the presence of extended black or birchen in his Silver Spangled Hamburgs. Carefoot (in press) also found a single gene difference between Gold Pencilled Hamburgs and Silver Spangled Hamburgs, the same varieties originally crossed by Punnett and Bailey (Punnett, 1923). Carefoot concluded from his study that these two varieties not only differed at the *E* locus, but that Silver Spangled Hamburgs carry melanotic (*Ml*), while the autosomal barred variety does not. Thus, he considered *Ml* to be the same gene that was previously described as *Sp*. He then crossed a Silver Spangled Hamburg male with double-laced Barnevelder females and again found spangling to depend on a single gene difference, in this case the addition of the *Db* columbian-like restrictor, since both parental stocks were *Ml*/*Ml*. Therefore, the genetic key according to Carefoot (in press) is the combined effect of *Db* and *Ml* which leads to the concentration of eumelanin into a spangle at the tip of the feather. It should also be noted that *Ml* has been reported to combine with *Co* to guide the formation of a single marginal lace (Moore and Smyth, 1972b). From the above results, Carefoot concluded that spangling is due to homozygosity for *E*, co^+ and the *Db-Ml-Pg* linked combination.

Spangling is also expressed on both extended black (*E*) and brown (e^b) backgrounds, the spangle being very small in the presence of e^b/e^b (Carefoot, in press). It appears that no one has isolated and studied the *E*-like allele present to see if it might not be birchen-like, and therefore more susceptible to secondary pattern modification. Another mutation that might conceivably be a modifier of the spangling pattern is mahogany (*Mh*), which has a spangling-like activity on the black breasts of wild-type males.

The down of the Silver Spangled Hamburg chick was referred to as 'marbled' by Hertwig (1933) and considered to be due to an autosomal recessive gene (*ma*), based on its relationship to black down. She reported that *ma* was linked to both the pea comb (*P*) and naked neck (*Na*) loci by approximately 33 and 46 crossover units, respectively. Based on the segregation for down color among the F_1 and F_2 progeny of the Spangled Hamburg x Barnevelder cross described above, Carefoot (in press) reported that down differences were consistent with the presence or absence of *Db*. Furthermore, linkage relationships between the genes in question have been estimated to be *P*-32-*Db*-10-*Ml*-10-*Pg*-26-*Na* (Carefoot 1987c, in press). Since map distances between *Db* and *Na* coincide with Hertwig's (1933) estimates for *ma*, *Db* appears to be identical to *ma*. However, Carefoot's assumption that the gold version of the downs of his Silver Spangled Hamburgs should look like the red-brown and cream-marbled downs of the gold-barred Fayoumi described by Moore and Smyth (1972c) is incorrect. The silver version of the gold-barred Fayoumi differed little from the gold and was apparent only in the cream spots on the back. The heads in both cases were red-brown, in the Fayoumi reported to be E^R/E^R *Co/Co Db/Db*.

According to Hutt (1949), Rittershaus in 1930 studied the inheritance of the head streak characteristically present in the down of Golden Spangled Hamburgs and concluded that it was due to a sex-linked recessive gene, *ko*. It is difficult to see why this is present in the gold and not the silver variety. As suggested by Hutt (1949), it may simply involve an interaction of s^+, the sex-linked gene that separates the gold and silver (*S*) versions of spangling in Hamburgs, although as he noted, Hertwig (1930) reported that a linkage study between *ko* and s^+ indicated them to be far apart on the Z chromosome. Whatever the reason for the down color difference, it would not be surprising to find it to be associated with the adult tail color difference between the golden and silver Hamburg varieties.

Mottling and pied. The secondary patterns mottling and pied have a related inhibitory effect on melanin deposition leading earlier workers to speculate that they might have a similar genetic basis (Asmundson and Milne, 1930; Hutt, 1949). Recent genetic evidence to support this contention (Carefoot, 1987d) warrants consideration of the mottling and pied traits together. The gene symbols *mo* and *pi* were assigned to mottling and pied respectively by Hutt (1949).

Mottling causes a white tip at the distal end of a certain proportion of the feathers, and is a characteristic of the Ancona and Mottled Houdan breeds (Figure 5.3h). On these self-black backgrounds, mottling appears on approximately one of every two to five feathers (Asmundson and Milne, 1930). More recently, Somes (1980) has shown that mottling is also responsible for the tricolor patterns (Figure 5.3i) including speckled, mille fleur and apical spangling (Old English Game type). The black down color of Ancona chicks, presumed to be *E/E mo/mo*, is greatly restricted ventrally. The remaining black pigment on the dorsal down led Schaible (1968) to consider the pattern as a transitory form of piebald spotting. In contrast, according to Somes (1980), *mo* does not have a discernible effect on noneumelanic down.

As described by Hutt (1949), the pied pattern of the Exchequer Leghorn has the general appearance of a very light Ancona. Like mottled feathers, some have black tips, while others are all black or all white. The Exchequer Leghorn is presumed to be *E*/*E* and the down color, when the pied genotype is added, resembles that of the Ancona.

Earlier investigators agreed that mottling and pied are inherited as autosomal recessives (see Hutt, 1949 and Somes, 1980 for review), except for Kimball (1953a) who considered a gene *As* to cause the apical white spangle in several tricolor varieties. Kimball proposed *As* to be dominant on an e^+ background, but recessive in the presence of *E*. A more recent examination of the dominance of mottling, however, further confirms its recessiveness and the acceptability of the *mo* symbol (Somes,1980).

Following a cross between a pied Exchequer Leghorn male and a mottled Ancona female, Carefoot (1987d) reported that both were caused by homozygosity for *mo*. He based this conclusion on an F_1 population of eight chicks, all of which developed plumage intermediate to the Exchequer and Ancona parents. He also proposed that since they were one and the same gene, the symbol *mo* should be retained. The distinct phenotypic differences between mottling and pied must then be attributed to modifying genes. Although numbers were small, no complementation occurred, so Carefoot's conclusion of a single gene is possibly correct; however, it is equally possible that the two traits are determined by different incompletely dominant alleles. The fact that Asmundson and Milne (1930) did not report any pied phenotypes among the progeny of a series of crosses between Anconas and other breeds, including the severely eumelanin-restricted Buff Wyandotte, suggests that the multiple allele hypothesis is the more plausible one. Asmundson and Milne (1930) did observe restricted phenotypes similar to pied, but these were only in the presence of sex-linked barring whose effects on plumage color have been well documented (Hutt, 1949; Cock, 1953; Silverudd, 1978).

The tricolor pattern of such varieties as the Belgian Mille Fleur bantam, Speckled Sussex, and Spangled Old English Game is characterized by a white tip, followed by an inverted V-shaped black band, with the rest of the webbed feather being buff or red-brown (Figure 5.3i). Surprisingly, its relation to *mo* remained unclarified until the report of Somes in 1980. Kimball (1953a) had speculated that his *As* gene was involved in both mottled and tricolor breeds. Earlier, Brumbaugh and Hollander (1965) reported that a cross between Speckled Sussex and Ancona produced only mottled offspring, suggesting that *mo* was involved in the speckled pattern.

Somes' (1980) study led him to attribute both the white tip and the black V-shaped band to the *mo* gene. In the presence of self-black (*E*), the base of the feather would be black as in the Ancona. In the presence of some black-inhibiting genotypes, the base would be nonblack resulting in the tricolor pattern. He suggested that in the Mille Fleur pattern the restrictor may be *Co* (columbian). The addition of silver (*S*) eliminated the pheomelanin from the base of the feather resulting in a white feather with a subapical inverted V-shaped band of black. Also of interest is the citation by Somes (1980) of a personal communication with Carefoot who indicated that the feathers of Anconas frequently show differences in the structural sheen between the black area near the white tip and the rest of the black feather. The distal black band was described as having a purple-black sheen, while the rest of the feather will have a greenish sheen. This suggests that in the tricolor pattern there is some relationship between pigment cell differentiation (eumelanogenesis vs. pheomelanogenesis) and the keratinous structure of the feather that leads to the differences in the purple and green sheens. Schaible (1968) had demonstrated quite convincingly that the melanoblasts of the Ancona have a retarded migration rate resulting in the very restricted black down pattern of this breed. It is tempting to hypothesize that this reduced migration rate may also delay the arrival of melanocytes from the dermal reservoirs to populate regenerating mottled feathers, leading to the amelanotic tip. The pigment cell migration fault may be in part due to the presence of *E* in Anconas, since *E*/*E* melanoblasts also have a slower migration rate in the absence of *mo* (Fox, 1949).

Sex effects on secondary pattern expression. An examination of the breeds and varieties exhibiting autosomal secondary patterns reveals frequent sex differences in expression (American Standard of Perfection, 1983). A major reason for this is the sexual dimorphic feather structure, which is estrogen-induced (see Hutt, 1949 for review). Sex differences in feather structure are removed by the hen-feathering gene *Hf*, which causes male plumage to be female-like. Actually this also involves estrogen, as *Hf* leads to an enhanced rate of estrogen synthesis in the skin (Somes et al., 1984). In addition to hormonal effects, specific plumage color genotypes are also involved, since differences in secondary pattern expression still occur between stocks with similar sexually dimorphic plumages.

Both Serebrovsky (1926) and Henderson (1956) attempted to explain homochromatic expression of feather patterns as being due to a single gene. Henderson's proposed *Hc* (for homochromous) was supposed to allow lacing expression in males in the absence of henny feathering. Although he seemed confused over the presence and the role played by henny feathering in nonhenny feathered phenotypes, his single dominant gene might have been *Co*, now known to be involved in the expression of lacing in the ventral plumage of the Silver Laced Wyandotte (Moore and Smyth, 1972b). In contrast, Kimball (1956a, 1956b, 1959) believed that his pencilling gene (*Pg*) was responsible for the absence of secondary pattern in males of pencilled breeds. Laced birds were believed to carry lacing, but not pencilling, and so they were laced in both sexes. The addition of his *Pg* to a single-laced genotype would again result in wild-type nonlaced males (Kimball, 1955). However, the stippled wild-type fails to express the secondary pattern in males. What is absent from both the stippled and pencilled genotypes is the columbian restrictor, *Co*, or a gene with similar activity.

On a non-*E* background, *Co* allows the expression of lacing in the ventral regions of male plumage, although expression of a completed lace also requires melanotic (*Ml*) (Moore and Smyth, 1972b). The eumelanin restrictor (*Db*) does not allow pattern expression in non-*E* males because it produces a black-tailed columbian phenotype in the male. In contrast, both *Co* and *Db* lead to secondary patterns on an *E*-like background in concert with genes in their particular interaction group. Examples include the laced Silver or Buff Polish and the barred Fayoumi (Smyth, unpublished). Therefore, the key to homochromosity of secondary pattern expression is the result of an appropriate multigene combination and the sex-hormone-conditioned feather follicle environment.

PHEOMELANIN EXPRESSION

Noneumelanized areas of wild-type plumage contain pheomelanins. Pheomelanic feather tissue may vary from dark mahogany-red to pale buff or be modified to distinct intermediate phenotypes, including the salmon coloration of female wild-type breast feathers and the body plumage of wheaten females. A popular alternative is to incorporate the silver (*S*) mutation, thus removing the pheomelanin and leaving an attractive black and white pattern. Many breeds recognized by the various Standards of Perfection have varieties that differ only in the presence or absence of pheomelanin, for instance Golden and Silver Laced Wyandottes, Buff and Light Brahmas, and Partridge and Silver Pencilled Plymouth Rocks.

In addition, due to the sex-linked inheritance of silver (*S*) and gold (s^+), these alleles are useful for commercial crosses to determine chick sex at hatching.

Sex-linked silver and gold. Silver and gold were studied as part of the early investigations of the application of Mendel's laws to animal genetics. Sturtevant (1912) and Davenport (1912) established that they were sex-linked, and that silver (*S*) was generally dominant to gold (s^+). The incomplete dominance of *S* over s^+ in males can at times make it difficult to identify the presence of *S*, as noted by Warren (1928). Since the degree of dominance of *S* appears to differ in different matings, it would appear to be highly influenced by modifying genes (Smyth, unpublished). Noting the inability of *S* to suppress pheomelanin, Brumbaugh (1971) referred to it as a 'leaky gene'.

Although gold (s^+) generally has been considered to be the gene responsible for red pigments, its specific role in pheomelanization is unknown. The appearance in S/W females of red pigment, sometimes to the point of almost masking the expression of silver, suggests that s^+ does not play a critical role in the synthesis of pheomelanin. The *S* locus may play a role in the availability of sulfhydryl groups, since Brumbaugh (1971) has proposed that silver (*S*) probably has an adverse effect on sulfhydryl incorporation during the terminal phase of pheomelanin synthesis. In an ultrastructural study of pigment formation in S/s^+ regenerating feathers, Brumbaugh (1971) also observed that *S* had no apparent effect on eumelanogenesis. Melanocytes were also present in normal numbers in the developing feathers from white areas. These pigment cells were melanogenically active and contained numerous simple and compound premelanosomes with little or no pheomelanin deposited on their matrices. That silver feathers do have a competent pigment system had been shown earlier by Henley (1947) and Cohen (1959).

A role for sex hormones in the development of silver and gold phenotypes was suggested by Nickerson (1946), who reported that castrated Silver Campine males deposited red pigment in regenerating feathers. Finlay (1925) had not observed such a change following castration of S/s^+ crossbred males. As pointed out by Brumbaugh (1971), Nickerson's study was confounded by the presence of the henny feathering phenotype prior to castration, followed by the postoperative cock-feathered plumage in which the pigment change was observed to occur. Studies by Groenendijk-Huijbers (1966, 1967), implicated both male and female hormones in the induction of pheomelanin. She found that pigment did not develop in embryos from a New Hampshire x Light Sussex cross following early gonadectomy. Her work verified the *in vitro* results of Hamilton (1940) who found that estrogen was able to restore the ability to produce pheomelanin in cultured pigment cells that otherwise produced only eumelanin.

A third allele, s^{al}, has also been proposed for the *S* locus (Werret et al., 1959; Cole and Jeffers, 1963). It is associated with the sex-linked imperfect albino trait, originally assumed to be at a different locus. The s^{al} mutation is reported to be recessive to *S* and s^+. Sex-linked imperfect albinism was first described by Mueller and Hutt (1941) and was found in several apparently unrelated Barred Plymouth Rock populations (Hutt and Mueller, 1943). Because of its albinotic effects on the phenotype, this mutation will be described in more detail in the section entitled 'White Plumage'.

Although the genetic data (Werret et al., 1959; Cole and Jeffers, 1963) strongly support the location of sex-linked imperfect albinism at the *S* locus, its residence there would appear to create strange bedfellows. The s^+ allele and its incompletely dominant allele *S* are expressed only in noneumelanized feather tissue, while s^{al} greatly reduces both eumelanic and pheomelanic pigmentation, with its greatest effect being on eumelanin. At the ultrastructural level, Brumbaugh's (1971) description of silver premelanosome structure differs distinctly from that reported for sex-linked albinism by Boissy et al. (1987). If Brumbaugh's contention that *S* is associated with sulfhydryl incorporation to switch the melanogenic pathway to pheomelanin production is correct, then a role for s^{al} is difficult to envision. Based on linkage relationship to common loci, Hutt (1960) located sex-linked albinism 1.2 crossover units from *S* which does not by itself invalidate the possibility that they could be alleles. Unfortunately, the matter cannot be resolved at this time, and available evidence favors the multiple allelic hypothesis.

Interestingly, silver is relatively ineffective in masking the salmon-colored pheomelanic pigments of wild-type and wheaten females. This explains the retained salmon breast in the female plumage of such silver stocks as the Silver-grey Dorking and Silver Duckwing Modern Game, as well as the striking sexually dichromatic plumage of the Salmon Faverolle. The addition of *Co*, and possibly *Db* and *Mh*, will alter the salmon pigment so that it is readily suppressed by *S* (Malone and Smyth, 1979).

Hypostatic silver is often found in self-colored black or white breeds, presumably in part due to its suppressive effects on pheomelanin. This is apparently less important in black breeds, but *S* is a standard component of the Barred Plymouth Rock genotype (see Hutt, 1949 for review). Although recessive whites may carry either *S* or s^+ (Quinn, 1936; Jeffrey, 1947), silver is an important part of the hypostatic genotype of dominant whites (Smyth, unpublished).

Silver and gold are readily expressed in columbian-like restricted downs, largely due to the presence of *Co* (Malone and Smyth, 1979). Neither is expressed on self-black *E* chicks, the creamy-white ventral regions of the down representing the delayed migration of eumelanoblasts (Fox, 1949), not an expression of *S*. Silver and gold can be differentiated without difficulty on wild-type (e^+), buttercup (e^{bc}) and probably speckled (e^s) downs, but with less certainty on brown (e^b), particularly the darker e^b downs. It is almost impossible to separate silver and gold on wheaten (e^{Wh} and e^y) down unless *Co* is present.

Differences in pheomelanin expression. Several single genes have already been cited that appear to dilute and/or change the appearance of pheomelanin. These were described in the section on columbian-like eumelanin restrictors and included the distinct colorations orange-gold and orange-tan, characteristic of *Co* and *Db*, respectively. Dilute (*Di*) has also been described as a dilution factor of pheomelanin, while mahogany (*Mh*) appears to darken the shade of red-brown, particularly in the presence of the wheaten (e^{Wh} and e^y) alleles at the *E* locus.

A major single gene inhibitor of pheomelanin is the autosomal recessive cream mutation *ig* (for inhibitor of gold). First analyzed by Taylor (1932b) and later by Punnett (1948), the cream dilution effect appears to be specific for pheomelanin with no apparent effect on eumelanin. Golden-buff feathers are diluted to pale buff and even mimic silver in some cases. Darker red plumages are also diluted, but the effect is less apparent. Wild-type males have straw-colored hackles and sickle feathers with silver expressed in the non-eumelanin areas of the secondary flight feathers. In general, males show a greater dilution of pheomelanin than females. The presence of cream could not be detected in the down on an e^+/e^+ background (Smyth, unpublished). Taylor (1932b) and Punnett (1948) differed in the way that they perceived cream to function. Taylor believed that cream dilutes the pheomelanins, while Punnett considered it to be the basic level that is intensified by sex-linked gold (s^+). Since the wild-type genotype does not include *ig*, Taylor's contention of a dilution action for cream appears to be the more acceptable.

It is not clear whether cream has a role of importance in standard breed and/or varietal plumage patterns. However, the author has identified cream in three different genetic stocks over the past 35 years. In one case, it appeared in a Light Brown Leghorn tester line that had been maintained as a closed flock for over 20 years, therefore appearing to represent a spontaneous mutation. The other two occasions were among the F_2 progeny from separate crosses, one involving silver barred Fayoumi, and the other a Golden Pencilled Hamburg obtained from a private breeder. Taylor's (1932b) original isolation was from Silver Spangled Hamburgs. Two of the three more recent recoveries were from silver varieties, while the Golden Pencilled bird came from stock also used to produce Silver Pencilled Hamburgs. This circumstantial evidence suggests that cream may be useful in certain silver breeds as an inhibitor of pheomelanin.

During their analysis of the plumage of a Buff Minorca, Brumbaugh and Hollander (1966) encountered another gene that inhibited pheomelanin. They called it champagne blond and suggested the gene symbol *Cb*, based on its apparent dominance. However, it appeared only in the heterozygous condition in a second backcross to red junglefowl, and then in only two individuals. Dominance was determined by the results of crossing one of the Cb/cb^+ birds back to wild-type. They suggested that *Cb* could be masked by their ginger gene (probably *Co*) and that the two were rather closely linked. Like cream, the champagne blond was not apparent on wild-type down. Adult males were said to resemble the golden duckwing pattern in the male, but the effects on female color were not presented.

Brief mention of another pheomelanin dilution gene was made by Kimball (1955) who isolated it from lemon-buffs and pale Golden Laced Wyandottes. It was recessive, like cream, but reported to be different.

An allelemorphic series that darkens the shade of pheomelanin was also proposed by Kimball (1955); however, no data were presented to substantiate its existence. Supposedly the most dominant allele, *Dk*, is present in Spangled Game, partridge varieties, and Dark Cornish, while Dk^1 is present in Dark Brown Leghorns. The most recessive allele, dk^+, was proposed to be present in the Indian subspecies of red junglefowl.

Studies to determine the genetic basis for intensity of pheomelanic pigmentation have attributed differences to multiple unidentifiable genes. Several of these involved the Rhode Island Red breed that varies in color from the very dark exhibition strains to the lighter and more variable production lines. Warren and Gordon (1933) made such a study and concluded that inheritance was by multiple factors, but that in general males were darker red in color than females. Hays (1935) found the F_1 progeny of a cross between dark and light red strains to be intermediate. In a later study utilizing optical density values determined from feather extracts, Hays et al. (1948) concluded that the dark coloration of exhibition strains was due to cumulative recessive genes, one probably being sex-linked.

A series of studies designed to study the inheritance and expression of pheomelanogenesis in three breeds, Rhode Island Red (RIR), New Hampshire (NH), and Buff Orpington (BO), representing dark, intermediate, and light levels of pigmentation respectively, was conducted by Somes and Smyth (1965a, 1965b, 1965c, 1966, 1967). They used optical density of whole feather extracts to estimate pheomelanic intensity for the three pure breeds, plus their F_1, backcross, and F_2 progeny. They reported that they were unable to identify individual gene effects, and therefore concluded that quantitative inheritance accounted for most of the pheomelanin differences (Somes and Smyth, 1965b). Differences between RIR and NH appeared to involve additive gene action, but the BO carried pheomelanin suppressor genes that were in part dominant or epistatic to pigment genes of the RIR and NH. These may have represented the dilute *(Di)* and champagne blond *(Cb)* genes found in Buff Minorca by Brumbaugh and Hollander (1966). When the extracts were further separated by the chromatographic procedures of Smyth et al. (1951), the brown melanin fractions, which make up the bulk of the pigment, again appeared to be inherited polygenically. However, the results suggested that separate genetic systems controlled quantitative differences for the trichochromes and the melanins (Somes and Smyth, 1965c).

In agreement with the observations of Warren and Gordon (1933), all male groups involved in the above studies were darker than the corresponding females, with the exception of the pure NH males (Somes and Smyth, 1965a). The presence of sex-linked slow feathering (*K*) was also found to be associated with darker pheomelanin color. In addition, estrogen levels were shown to reduce pheomelanin density by as much as 57 percent in lighter colored birds (Somes and Smyth, 1967). This may explain the antagonism between early sexual maturity and dark red color reported by Hays (1932), whereby adult plumage in pullets would develop under the influence of higher levels of estrogen in earlier maturing birds.

Down color variations have also received attention, particularly those involving the presence of dark head spots of variable sizes and faint back striping on RIR downs (see Hutt, 1949 for review). In general, chicks with such markings are more likely to be females, but not with a sufficient degree of accuracy for practical sex separation (Warren, 1929; Byerly and Quinn, 1936). Warren reported little relationship between down color intensity and surface color in adults. However, Somes et al. (1966) recalculated his data and found a positive relationship. Using chicks from the above-mentioned study involving RIR, NH and BO, Somes et al. (1966) found very high and significant correlations between down color scores and optical densities of postjuvenile feather extracts, suggesting similar genetic determination.

GENERAL DILUTION FACTORS

Several mutations that result in the dilution of melanins have already been described. These primarily affect pheomelanin, as in silver, cream, dilute, and champagne blond. Others affecting only eumelanin, or both, will be considered in this section (Table 5.4).

Blue. Bluish-slate plumage is a characteristic of the Blue Andalusian breed and it also occurs in Blue Orpingtons, Crested Blue Polish, and several varieties of Old English Game bantams such as Self Blue, Silver Blue and Lemon Blue. Females of the standard-sized breeds are characterized by black marginal lacing on otherwise blue feathers throughout the plumage. Males are similarly laced except in the sexual dimorphic regions (neck, back, saddle, and wing bow) which are black or very dark blue. In contrast, some blue phenotypes, like that of the Self Blue Old English Game bantam are essentially bluish-slate without black lacing.

Blue pigment is a modified eumelanin as indicated by solubility and light absorption properties (Bohren et al., 1943). Additionally, only black melanin is modified by the blue mutation as evidenced by blue wild-type and columbian patterns. Blue melanosomes differ from black in that they are spherical rather than rod-shaped (Lippincott, 1918; Bohren et al., 1943). Melanosome distribution in the keratinous structure also varies, with those in black feather tissue being deposited throughout the barbs and barbules, while in blue areas the pigment granules are clustered or loosely clumped. A further difference is that the proximal barbules are more heavily pigmented in blue feathers, the reverse being the case for black feathers. These differences can be observed in the black and blue areas of the same black-laced blue feather.

The fact that blue chickens do not breed true was explained by Bateson and Punnett

Table 5.4. Genes that have a general dilution effect on the plumage.

Name	Symbol	Mode of inheritance[1]	Phenotypic effect
Blue	*Bl*	Incompletely dominant	*Bl*/*bl*$^+$ is blue; *Bl*/*Bl* is blue-splashed white
Lavender	*lav*	Recessive	Dilutes both eumelanin and pheomelanin
Pinkeye	*pk*	Recessive	Dilutes both plumage and eye color
Red-splashed white	*rs*	Recessive	Dilutes all melanin, particularly eumelanin
Grey	none	Recessive	Dilutes black to brownish-grey
Erminette	none	Incompletely dominant	May be an allele at *I* locus

[1] All are autosomal mutations.

(1906), when they showed that blue chickens were heterozygous for an incompletely dominant mutation (later assigned the symbol *Bl*). As confirmed by Lippincott (1918), blue x blue matings result in a ratio of 1 black:2 blue:1 blue-splashed white. Homozygous *Bl* birds are essentially white with a few blue markings, their number and size being reduced by other genes that restrict eumelanin expression, such as sex-linked barring and mottling (Smyth, unpublished). The gene action that results in blue ticking is not known. However, it should be noted that small black ticks occur within the blue feather areas (Lippincott, 1923; Cock, 1953), and although less obvious, within the blue splash of *Bl*/*Bl* feathers (Lippincott, 1923). Based on heterokaryon analysis of the genetic control of melanin synthesis, Wilkins et al. (1982) suggested that *Bl* is a structural gene for the premelanosome.

It is an accepted fact that many commercial White Leghorn lines carry hypostatic blue. In addition, the author has uncovered blue in two commercial white meat lines. Whether this is simply an indication of common backgrounds, or due to the pigment suppressing effects of blue-splashed white (*Bl*/*Bl*), or both, is not known.

The genetic basis for the difference between the Andalusian type of blue-black distribution and the self-colored blues has received little attention. Carefoot (1988) concluded that the laced pattern of the Andalusian bantam was due to homozygosity for the eumelanin intensifier, melanotic (*Ml*), the pattern gene (*Pg*), and the columbian restriction gene (*Co*), on an *E*/*E Bl*/*bl*$^+$ background. Cote (1976) noted that blue was ineffective in modifying black melanin attributable to *Ml* and *Lg*, which suggests that the absence of these genes might result in a self-blue pattern. The difference is probably not due to *Co*, since Cote (1976) recovered a *Co*-like gene from a self-blue male from a random-breeding meat-type population. The author later crossed similar self-blues to the heavily eumelanized Massachusetts Recessive Black (RB) line, then mated the darkest F_1 offspring *inter se* and recovered the black-blue Andalusian pattern in the F_2. It was not determined whether or not this was due to melanotic (*Ml*) and the secondary pattern (*Pg*) genes alone, since the RB line has additional eumelanizers. The net effect of the recessive black complex is to convert the sexually dimorphic nonblack plumage of wild-type males to self-black. This area coincides with the eumelanized area of the Blue Andalusian male plumage.

Lavender. A true breeding blue dilution occurs in the Porcelain variety of the tricolor Mille Fleur and was found to be due to an autosomal recessive mutation *lav* (Brumbaugh et al., 1972). Lavender dilutes both eumelanin and pheomelanin, the former to a light grey and the latter to a buff or cream. Chick down is also diluted so that on a wild-type background the chick is grey, with the eye and back stripe still visible. Somes (1980) verified the presence of *lav* in the Porcelain Mille Fleur. He also described its dilution effects on the basic tricolor pattern, which he attributed to the combination of mottling (*mo*) and a columbian dilution factor (probably *Co*).

The *lav* gene appears to cause pigment dilution in feathers because of the inability of melanocytes to transfer their pigment granules into the keratinocytes of the developing feather. Lavender does not appear to affect melanin synthesis, so that the cytoplasm of the cell body is congested with melanosomes. The dilution effect is due to the appearance of the pigmented melanocytes surrounded by essentially colorless keratinocytes. Brumbaugh et al. (1972) first reported that the lavender effect on pigment granule transfer was due to abnormal dendrite formation; the dendrites were either very short or absent. However, in a later study, Mayerson and Brumbaugh (1981) showed that dendrites are present, and that the defect is associated with a defect in melanosome translocation between the perikaryon and the tip of the dendrite. However, no defect of the intracellular filament network was apparent ultrastructurally.

Grey. An autosomal recessive mutation that diluted the black pigment of Black Australorps to a light to dark brownish-grey was described in a brief report by Erasmus and Liebenberg (1958). In older birds, the color bleached to a dirty yellow. It was noted that one bird became depigmented; however, this may be unrelated to the grey mutation since cases of spontaneous amelanosis had been previously reported in the Black Australorp breed in South Africa (Gericke, 1934). Smyth (1989) has pointed out that the South African cases of depigmentation closely resemble the Smyth chicken, a model for autoimmune amelanosis.

Pinkeye. The pinkeye mutation was first described by Warren (1940) after its appearance in a flock of White Plymouth Rocks. A cross between these pinkeyed birds and a heterozygous autosomal albino (C/c^a) resulted in normal eye and feather color in the F_1, indicating that the two pinkeye types were due to different mutations. Subsequent results indicated that pinkeye was due to an autosomal recessive gene *pk*. When homozygous it diluted black melanin to a blue-grey color, but appeared to affect only eumelanin. What would appear to be the same mutation was later isolated from another White Plymouth Rock flock by Roberts et al. (1952). They called their mutation 'red eye', to distinguish it from the pink eye of true albinos. Warren (1940) noted earlier that eye color was darker in *pk/pk* birds with colored plumage than those that were recessive white. Both pinkeye and red eye were reported to result in impaired vision.

Brumbaugh (1968) showed that pinkeye birds have normal tyrosinase activity and their premelanosomal matrix appears similar to that of controls. However, *pk* retards the attachment of the eumelanin polymer to the premelanosome. It was this characteristic that allowed Brumbaugh to describe the chicken premelanosomal structure in detail. Brumbaugh and Lee (1975) later demonstrated ultrastructurally that *pk* affects both retinal pigment epithelial cells, which arise from the neural cup in the early embryo, and the ocular and cutaneous eumelanocytes of neural crest origin. They suggested that *pk* is a structural mutation that affects melanin binding to the premelanosome. However, the role for the *pk* locus was later altered, based on *in vitro* complementation tests between several mutations that reduce or block pigmentation (Wilkins et al., 1982). Heterozygotes, where two mutants were present in the same nucleus, were compared with heterokaryons, where two mutants were present in separate nuclei within a single cell. Their results with *pk* led them to conclude that the *pk* locus plays a major role in normal premelanosome synthesis or assembly, but as a nuclearly restricted regulatory element.

Red-splashed white. A major melanin inhibitor, red-splashed white, segregated from a flock of Rhode Island Reds and was found to be due to an autosomal recessive gene by Quinn (1934). Assigned the gene symbol *rs* by Hutt (1949), mutant chicks were white with a red head spot, while adults were white with variable red splashes. When crossed with Black Jersey Giants and Light Sussex, *rs* proved to be an even more effective inhibitor of eumelanin (Quinn, 1935). Some *rs*/*rs* blacks were totally white, while others showed black spots on the head, and limited ticking on the body plumage.

Unfortunately, this mutation has not been maintained, nor has it been reidentified. This is unfortunate since its suppressive effects on both eumelanin and pheomelanin would have made it a valuable asset for studies on the genetic control of pigmentation.

Erminette. The attractive pattern of the Erminette fowl, has been described and analyzed by Hutt (1964). Descriptions and pictures suggest the pied secondary pattern, that is, variable black and/or white feathers, often black with white tipping or lacing. Chick down also shows black spots and flecks on a cream background, not unlike an exaggerated expression of heterozygous dominant white in the presence of *E*. Beaks and shanks are also spotted with black at all ages. No information was offered on the effect of erminette on pheomelanin.

Genetic tests showed that erminettes are always heterozygous and appear to have been *E*/*E*, since they are black in the absence of the erminette factor. Homozygous mutants are pure white, thus similarity to dominant white is again suggested. Unfortunately, no matings to dominant white (*I*) were made, so a possible allelic relationship with *I* was not determined. The mutation would not likely reside at the *mo* locus, since mottling and pied are both simple recessives, while erminette is incompletely dominant.

Partial feather achromatosis. A partial feather achromatosis that diluted both eumelanin and pheomelanin was described by Washburn and Smyth (1967). Expressed on columbian-like restricted patterns, it appeared in a newly synthesized columbian restricted (*Co*/*Co*) line that originated from a White Plymouth Rock x Light Brown Leghorn cross. The grey back down coloration of the *Co*/*Co* line was diluted to a light grey. Although both pheomelanin and eumelanin were inhibited in subsequent plumages, the dilution effect was much greater for pheomelanin. In males, essentially all pheomelanin was eliminated and the s^+/s^+ males looked like silver homozygotes. In females, the normal red-brown color was diluted to buff, and small amounts were present on most body feathers which were otherwise white. They concluded that the achromatosis was due to a recessive mutation *mi*, that modified the *Co*/*Co* pattern on either an e^+/e^+ or e^b/e^b background.

Subsequently, Smyth and Malone (1979) found that the *mi* of Washburn and Smyth (1967) was the *Db* columbian restriction gene. They showed that homozygous *Db* and *Co* interact to result in the partial achromatosis described above. Genotypes containing both mutations (Db/db^+ Co/co^+; Db/db^+ Co/Co) show intermediate degrees of achromatosis in a dose-dependent manner. Interestingly, either homozygote alone on a e^b/e^b (brown) background shows some achromatosis, in the saddle feathers of *Db*/*Db* males and in the flight and coverts of *Co*/*Co* males. Additional evidence that *Db* and *mi* are the same was provided by linkage data. Washburn and Smyth (1967) reported that *mi* and *P* (pea comb) were 28 crossover units apart. This agrees well with a map distance of 32 units between *Db* and *P* recently found by Carefoot (1987c).

Light down. According to Hutt (1949), Hertwig and Rittershaus in 1929 described a dominant sex-linked gene that diluted brown down to a whitish-yellow. It was found to be carried by Barred Plymouth Rocks so it apparently did not affect black melanin in the down. No adult descriptions were presented, except that at least some chicks with light down developed into adults with a considerable amount of gold and brown in the plumage. This mutation does not appear to have been described again.

WHITE PLUMAGE

White plumage (Table 5.5) may be accompanied by varying degrees of melanization of other tissues. Ocular melanin may be absent, greatly reduced, or unaltered, while skin and internally melanized tissue may or may not be affected. White-plumage phenotypes involving the absence or marked reduction of ocular melanins qualify as albinos, which can be either tyrosinase negative or positive (see Witkop, 1983 for review). In contrast, there are no examples of ocular amelanosis that do not affect feather pigmentation, although the previously described pinkeye mutation appears to have a greater effect on eye than feather pigmentation. A heritable spontaneous postnatal loss of feather melanization is also accompanied by the elimination of uveal tract and epidermal melanin (see Smyth, 1989 for review).

White feathers are economically desirable to the commercial poultry meat industry because they leave no residual melanin in the follicle after feather removal. This makes it feasible to process poorly feathered and/or pinny birds without penalty.

Dominant white. The dominant white mutation, *I*, is a breed characteristic of White Leghorns and most current commercial meat stocks that have been synthesized for that particular purpose. Dominant white is also an important contributor to several patterned breeds including the White Laced Red Cornish, Buff Laced Polish and both the Old English and Modern Red Pyle Games. In addition, *I* may be present in certain recessive white breeds and varieties, including White Minorca, White Wyandotte and White Dorking.

One of the first animal traits to be investigated following the rediscovery of Mendel's classical work, dominant white was shown to be a dominant mutation by Bateson (1902). A number of investigators verified this work, and the mutation was assigned the gene symbol *I* by Hurst (1905). *I* is incompletely dominant to its recessive allele i^+, which is associated with the absence of the *I*-effect on pigment. However, the phenotypic effects of *I* depend on its epistatic effects on melanin, primarily eumelanin. Most of the early studies were done with White Leghorns, which are, in most cases, black barred silver (E/E B/B S/S) birds carrying dominant white. On a self-black or barred background, I/i^+ chicks and adults are either black-flecked white or light greyish in down or feathers. The amount of black spotting is reduced when sex-linked barring (*B*) is present. In contrast, *I* is a relatively ineffective inhibitor of pheomelanin. It is undoubtedly for this reason that White Leghorns carry *E* to mask red pigment and *S* to remove it from any nonblack areas.

The difference between red and black pigment suppression by *I* has resulted in some interesting plumage phenotypes. The red-pyle patterns result from the removal by *I* of the eumelanin, but not the pheomelanin, from wild-type plumage. Thus, red-pyle females have salmon breasts, while males have gold or red pigment in their hackles, wing bow and bay, back and saddle feathers. The white areas can be increased or reduced by selection to alter the presence of eumelanin, as in birds with white vs. pheomelanic hackles. Dominant white is also the key to the white-laced red or buff patterns of the White Laced Red Cornish

Table 5.5. Genes that result in white, or essentially white plumage color.

			Phenotype of homozygote	
Name	Symbol	Mode of inheritance	Plumage	Eye
Dominant white	I	Incompletely dominant	White	Pigmented[1]
Dun	I^D	Incompletely dominant	"Whitish"	Pigmented
Recessive white	c	Recessive to C^+	White	Pigmented[2]
Red-eyed white	c^{re}	Recessive to C^+, c	White	Dark red
Recessive albino	c^a	Recessive to C^+, c, c^{re}	White	Pink
Imperfect albino	s^{al}	Recessive to S, s^+[3]	Partially pigmented[4]	Light red

[1] Bay eye, iridial and retinal pigment epithelium melanized.

[2] Usually bay-eyed, but can be part or all brown with appropriate genotypes.

[3] Only sex-linked mutation of group, allelic to silver and gold.

[4] Black plumage becomes greyish-tan and red plumage becomes light red.

and the Buff Laced Polish, respectively. These are otherwise black-laced reds or buffs with *I* added to remove the outer black lace from each feather. Interestingly, dominant white has also been found in some buff breeds where eumelanin in the columbian restricted areas is undesirable (Hutt, 1949).

The gene action of dominant white that results in pigment suppression has interested a number of investigators. That *I* is an autonomous mutation was shown by a number of investigators who utilized it in embryonic tissue exchanges (see Hutt, 1949 for review). Pigmented donor tissue consistently resulted in large colored grafts on the dominant white host, although the reverse exchanges resulted in smaller white grafts, probably due to migration of host melanoblasts into the graft tissue. Developing feathers from White Leghorns were shown by Dorris (1938) and Hamilton (1940) to have melanocytes containing pigment. Willier and Rawles (1940) reported that the epidermal collar of developing feather germs contained normal eumelanin-producing melanocytes, but that these round up and die prior to depositing more than a few melanosomes. White Leghorn pigment cells were also reported to have a life span of only four days in culture in comparison to a ten-day survival for cells from the Barred Plymouth Rock (Hamilton, 1940).

Ultrastructural examination of regenerating feather germs by Brumbaugh (1971) indicated that the dominant white melanocytes of White Leghorns contained irregularly shaped and assembled melanosomes. An extensive study of pigment cell activity in the same breed by Jimbow et al. (1974) led them to conclude that the hypomelanosis results from an inhibition of melanin synthesis and transfer to the feather keratinocytes followed by early cell death. They found *I/I* pigment cells to contain aggregates of membrane-bound melanosomes in the process of degradation. They further suggested that the formation of these acid phosphatase-containing autophagosomes, and their fusion into larger bodies, led to early death of the pigment cell. Therefore, it appears that *I* is responsible for a melanocyte defect that leads to the formation of reduced numbers of irregularly assembled melansomes. Apparently, the melanocyte attempts to protect itself from these abnormal, and likely cytotoxic, organelles by packaging them for degradation in autophagosomes, ultimately leading to cell necrosis and reduced melanin in the keratinized feather.

Interestingly, Brumbaugh and Lee (1975) concluded from their ultrastructural studies that *I* contributes a structural component involved in premelanosome formation. Later, Wilkins et al. (1982) concluded from their heterokaryon-heterozygote comparisons that *I* is a nuclear-restricted regulating element.

Since dominant white affects pigmentation of the feather and choroid of the eye, but not the retinal and iridial pigment epithelia, it appears that *I* affects only melanocytes of neural crest origin (Brumbaugh and Lee, 1975). However, both feather eumelanocytes and pheomelanocytes are of neural crest origin with the latter being only slightly inhibited by *I*. Brumbaugh (1971) hypothesized that dominant melanin deposition might lead to the autophagocytosis reported by Jimbow et al. (1974). Since eumelanocytes produce more melanosomes, containing greater amounts of melanin, than do pheomelanocytes (Brumbaugh, 1971), this could explain why pheomelanization escapes much of the *I* inhibition. In either case, dominant white expresses its pigment effects in a dose-dependent manner; that is I/I inhibits either eumelanin or pheomelanin synthesis more than does I/i^+.

A new allele, found in a Pit-Gamecock, has been proposed for the *I* locus by Ziehl and Hollander (1987). Like dominant white, the effect of this mutation is on eumelanin. The heterozygote modifies black pigment resulting in uniform brownish coloration, hence the name 'dun' and the proposed symbol I^D. Homozygotes are described as whitish. Although there are some similarities to pinkeye (*pk*), the eyes of duns are pigmented. Heterozygous dun chicks (I^D/i^+) resemble blue (Bl/bl^+) in the down, but this color is lost in subsequent plumages. Allelism was based on the results of dun x dominant white and testcross matings. Although the authors do not indicate the dominance relationships of *I* and I^D, dun was described as whitish or near-white and two I/I^D individuals as white, thus *I* would appear dominant to I^D.

The *c* locus. Although the *c* locus of the fowl is now known to be multiple allelic (Brumbaugh et al., 1983; Smyth et al., 1986), the mutant phenotype referred to as recessive white is the most prevalent among the white breeds and varieties that do not carry dominant white. Recessive white is a varietal characteristic of Plymouth Rock, Wyandotte, Minorca, Orpington, Jersey Giant, Dorking, Langshan, Silky and other breeds.

Recessive white (*c*) was another of the earliest animal traits to be studied during the first investigations of Mendel's principles. Its autosomal recessiveness was established by Bateson and Punnett (1906) and verified by a number of early workers (see Hutt, 1949 for review). Its epistatic effect over other pigment genes was also demonstrated, and common hypostatic genotypes carried by recessive white stocks were analysed by Quinn (1936) and Jeffrey (1947). Their results indicated more segregation at the *E*, *B*, and *S* loci than was the case for the White Leghorns. This suggests that recessive white needs less assistance than does dominant white to initiate pure white plumage.

One puzzling aspect involving the inheritance of recessive white was the cross between White Silkies and several other recessive white breeds that reportedly resulted in colored F_1 progeny and an approximate ratio of 9 colored:7 white in the F_2 (Bateson and Punnett, 1908). This was explained as an example of complementary gene action, whereby two dominant genes need to be present for melanin pigmentation. One of these was hypothesized to provide the necessary substrate or chromagen (*C*), and the other, an enzyme or oxidase (*O*). However, no one appears to have since confirmed these results, although the original breeds have been used. Hutt (1949) suggested that the Silky might have carried dominant white, which is credible when one considers that the progeny were described as colored or light, including some that were buffy. As pointed out earlier, dominant white is sometimes carried by recessive whites. In contrast, it is possible that some long lost recessive mutation affecting melanization did exist, or possibly may still be present in an untested white stock.

A third allele at the *c* locus was added when Brumbaugh et al. (1983) reported that autosomal albinism, previously considered to be a recessive mutation at a separate locus (Warren, 1933), was actually an allele of recessive white. He suggested the symbol c^a. However, the recessive white utilized by Brumbaugh et al. (1983) had dark red eyes that were readily detectable by shining a light through the heads, but not normally apparent without such back-lighting. Since light would not pass through the fully melanized eyes of a number of other recessive white lines, the allelic make-up of the *c* locus was further investigated by Smyth et al. (1986). Their study indicated that the dark-eyed recessive white gene was allelic and dominant to the red-eyed type. The symbol *c* was retained to represent the more common dark-eyed phenotype, white c^{re} was suggested for the red-eyed allele. The order of dominance for the four alleles is $C^+>c>c^{re}>c^a$. The wild-type C^+ allele appears to be completely dominant to all the others. However, Carefoot (1979) has observed that C^+/c heterozygotes can be identified when segregation is on an inbred background in the Partridge Wyandotte. The effect of the heterozygote was to dilute pheomelanin. In fact, the *C* locus in general appears to inhibit pheomelanin more effectively than it does eumelanin, as it also does in mammalian systems (see Searle, 1968 for review). Recessive white appears to be completely dominant to c^{re} and c^a, while the relationship between c^{re} and c^a is one of incomplete dominance (Smyth et al., 1986).

Down colors associated with homozygosity for the three mutants, *c*, c^{re}, and c^a, indicate that all but c^a/c^a can produce some melanin in the down. Recessive white (*c*) down varies most with some chicks being almost as dark as light colored Barred Plymouth Rock males. In other cases, *c*/*c* chicks have amelanotic down. The hypostatic genotypes that are associated with these down color variations have not been clarified; however, the black extension factor, *E*, is present when the downs are greyish (Jaap, 1943). The intensity of the grey color appeared to be due to modifying factors, although female chicks were found to be on the average darker than males. Less is known about the red-tinted and dusky or dirty greyish-yellow downs, although in two populations tested by the author, they were not associated with *E*. Genotypes responsible for pure white down have not been isolated, although in some cases they can be attributed to the combination of hypostatic *I* and *E*. Downs of c^{re}/c^{re} chicks may be either white or slightly tinted with grey.

Adult plumages of all three *c* mutants are typically white, even though the chick down may have been melanized. The recessive whites (*c*) may show some pigmentation, usually light greyish. Such birds are found in segregating and synthetic populations derived from crosses among commercial meat stocks which tend to carry strong eumelanin intensifying factors. One such line that carries strong shank and eye melanization, also has a very high incidence of eumelanic somatic feather mutations, to the extent that they resemble heterozygous dominant whites carrying *E* (Smyth and Boyle, unpublished).

Ultrastructural and cytochemical studies by Brumbaugh et al. (1983) of both ocular and feather melanocytes of red-eyed (c^{re}) and albino (c^a) chicks, suggested that both are cytochemically tyrosinase negative. Boissy et al. (1987) came to the same conclusion in a similar analysis of the albino (c^a) phenotype. Melanocytes were observed to be attempting to make melanosomes but these were few in number and incomplete, causing hypertrophy of the Golgi system. In addition, numerous large premelanosomic vesicles, containing what appeared to be lattice-like matrix material, were present (Brumbaugh et al., 1983; Boissy et al., 1987). As pointed out by Brumbaugh et al. (1983), the retinas of c^{re}/c^{re} birds contain a small amount of pigment. Surprisingly, they also found a few pigmented melanosomes in the retinas of older c^a/c^a birds, and therefore, these albinos are not totally devoid of melanin.

At the present time it is not possible to conclude with certainty whether the *c* alleles represent mutations of the structural tyrosinase gene, or exert their effects on the enzyme in a regulatory function. Oetting et al. (1985) have shown that both c^{re} and c^a produce electrophoretically and antigenically tyrosinase-like molecules that are, however, inactive. They suggest that the *c* alleles may represent an amino acid substitution in an active site, although post-translational modifications cannot be ruled out for these mutations. In any event, *c*, and to lesser extents, c^{re} and c^a, are 'leaky' genes. This not only involves specific tissue differences, but is also expressed in plumage, particularly in the down.

Sex-linked imperfect albinism. Although the genetics of sex-linked imperfect albinism (s^{al}) was discussed previously in the section on sex-linked silver and gold, it is included here in order to compare its albino-like phenotype with dominant white and the *c* alleles.

Sex-linked albinism was designated 'imperfect' by Mueller and Hutt (1941) because it did not completely eliminate pigment in the Barred Plymouth Rock where it was first found. Such mutants were described as ghost-barred. They reported that down colors varied from almost pure white to dusky chocolate. In a population designed by the author to segregate for s^{al} and its allele s^+, the albino phenotype on an *E* background is a light greyish tan in both down and adult plumage, while on a red columbian background, the down is creamy colored and adult feathers are tinged with red pigment. Mueller and Hutt (1941) observed limited numbers of pigment granules in albino feathers, while Boissy et al. (1987) found s^{al} melanocytes to be positioned normally in the barb ridges of regenerating feather germs. Eye color of imperfect albinos varies from light pink to darker reddish shades depending on the genetic background. The lightest pink appears in the presence of dominant white (*I*). Darker shades, which further darken with age, occur in normally pigmented birds (Mueller and Hutt, 1941). These findings were verified by histological observations. The $s^{al}\,I$ combination was found by Champion (1958) to have reduced amounts of ocular melanin, and this was limited to the pigment epithelium of the ciliary body and the iris. In nondominant white birds the retinal pigment epithelium was also melanized, although at reduced levels when compared to control eyes (Mueller and Hutt, 1941).

Since some melanin synthesis occurs in the presence of homozygous and hemizygous s^{al}, it is not surprising that such birds are tyrosinase positive. Yamamoto and Brumbaugh (1984) reported that s^{al} cells in culture produce approximately twice as much tyrosinase with about 30 percent higher specific activity than normal pigment cells. In contrast, Boissy et al. (1987) found only control levels of tyrosinase in s^{al} cells. The latter also found that in contrast to the situation in control cells, a lower-molecular-weight form was more abundant than was a higher-molecular-weight protein. Ultrastructural examination indicated that the cytoplasm of albino melanocytes contain a few morphologically normal premelanosomes, and numerous abnormal ones. Boissy et al. (1987) further suggested that tyrosinase and acid phosphatase may not be efficiently transported to the premelanosomes, but may be misdirected to some other membrane-bounded compartment.

Hypomelanic pleiotropies. Deleterious pleiotropic effects have long been associated with many of the genes that markedly inhibit melanization in a variety of species, including man and other mammals (see Witkop, 1983 and Searle, 1968 for review). Probably the best characterized example in chickens involves the autosomal recessive albinism (c^a), which Warren (1933) noted was associated with very poor eyesight and photophobia. More detailed studies (Pardue et al., 1985; Pardue and Smyth, 1986) have shown that the c^a/c^a genotype is also associated with a number of other statistically significant pleiotropic effects involving embryonic and early postnatal development; these include shorter down, increased incidence of yolk sac protrusion, subcutaneous hemorrhages and inflammation, increased late embryonic and postnatal mortality to eight weeks of age, reduced postnatal growth rate, and smaller bursal weights. These factors, plus a greater retention of yolk sac contents and reduced levels of plasma glucose and protein at hatching, suggested that autosomal albinism affects certain fundamental mechanisms that regulate development. Additionally, the close linkage between recessive white and an embryonic lethal reported by Dunn (1923) might have been the result of another mutation at the *c* locus, whose phenotypic effects on embryonic development were more severe than those of c^a. Such an hypothesis could explain the shortage of white chicks among Dunn's backcross progeny. The source of recessive white for these matings was the White Wyandotte variety.

The recessive white allele (*c*) has also been shown to have a small but consistent depressing effect on growth rate (see Chapter 20, and Fox and Smyth, 1982 for review). In contrast, the unrelated dominant white mutation (*I*) has not been found to consistently affect growth rate (see Chapter 20, and Smyth, 1968 for review).

A number of similarities between the pleiotropic effects of c^a and sex-linked imperfect albinism (s^{al}) can be found in the literature. Short down on imperfect albino chicks was noted by Mueller and Hutt (1941) and confirmed by Champion (1958). In addition, s^{al} is associated with many of the developmental defects of the embryo and neonate that are listed above for autosomal albinos, including reduced early postnatal growth rate (Silversides and Crawford, 1986, 1987). This similarity is surprising, since c^a and s^{al} must exert their influence on the melanization process differently, since c^a is a tyrosinase negative mutation, while s^{al} is tyrosinase positive (Brumbaugh et al., 1983; Yamamoto and Brumbaugh, 1984; Boissy et al., 1987).

Due to the above similarities between c^a and s^{al}, Smyth et al. (1987) compared a number of hypomelanic phenotypes, including those associated with the *c* alleles (*c* and c^a), s^{al}, *I* and *pk*. All of these were tyrosinase positive except for c^a. All comparisons were made using randomly segregating populations, and were between mutant and normal sibs. The c^a and s^{al} associations reviewed above were confirmed, while surprisingly, pinkeye (*pk*) had similar relationships even though these birds have considerable feather pigmentation. Recessive (*c*) and dominant (*I*) whites showed similar but statistically nonsignificant trends. These data indicate that mutations causing albino or albino-like phenotypes result in similar pleiotropic effects on embryonic and early postnatal development. This appears to be due to the absence of melanin or functional melanocytes, since the mutations included appear to disrupt the melanization process at different stages. Since the sex-linked albino and pinkeye phenotypes have more feather melanin than do recessive and dominant whites, but share the ocular hypomelanic characteristic with c^a/c^a albinos, the loss of eye melanin appears to be more closely associated with development than does loss of feather melanin. The less severe pleiotropies associated with the recessive white and dominant white phenotypes cannot be attributed to a protective buffering effect of accumulated beneficial modifiers since the populations studied were segregating on diverse outcrossed backgrounds.

Spontaneous postnatal amelanosis. A number of reports have described normally pigmented chickens that subsequently developed partially or totally amelanotic plumage (see Hutt, 1949 for review). Causative factors appeared to include neoplasia and endocrine disorders, while several cases suggested unknown etiologies with a genetic basis.

According to Hutt (1949), a crossbred black pullet was observed to become 75 percent amelanotic by Finch in 1909 and then to produce a totally amelanotic daughter. In 1938, Gericke reported the spontaneous occurrence of two totally amelanotic Black Australorp females that subsequently produced amelanosis among their F_1 and F_2 progeny. More recently, Smyth et al. (1981) described a line, derived from a single spontaneous amelanotic female, that has a high (80-92 percent) incidence of feather amelanosis. The defect is first apparent between five and 20 weeks of age. This line, originally referred to as the DAM (delayed amelanotic) line, has since become known as the Smyth line, following its general acceptance as an animal model for human vitiligo (see Smyth, 1989 for review).

Of the above cases of vitiligo-like postnatal pigment loss, only the amelanosis of the Smyth line has received much study (see Boissy and Lamoreux, 1988 and Smyth, 1989 for review). The loss of ability to melanize feathers has been shown to be due to an inherent pigment cell defect that appears to be related to a hypermelanic activity (Boissy et al, 1983; Boissy et al., 1986). Interestingly, selection for darker brown (e^b/e^b) down within the line has also been shown to increase the incidence of amelanosis (Boyle et al., 1987). Abnormal melanosomes lead to the formation of autophagosomes and eventual cell necrosis (Boissy et al., 1985). This process is followed by an immune response that eliminates the abnormal melanocytes, and subsequently, differentiating melanoblasts moving from their dermal reservoirs to newly regenerating feather germs. That undifferentiated melanoblasts are not destroyed by immunocytes is shown by periods of temporary remelanization that occur in females during postlay feather molts (Smyth et al., 1985).

Smyth line amelanosis also affects uveal tract and skin pigment. In approximately 50 percent of birds with extensive feather amelanosis, an immunogenic chorioretinal disease, leading to blindness, is triggered by the inflammatory reactions associated with destruction of the choroidal melanocytes (Fite et al., 1983; Boissy et al., 1983). The line is also characterized by lower incidences (less than 3-4 percent) of autoimmune thyroiditis, and an alopecia-like feathering defect that also involves an immune response (see Smyth, 1989 for review). Therefore, the Smyth line also serves as an avian model for autoimmune disorders in general.

The mode of inheritance of the Smyth line amelanosis appears to involve several genes plus lesser modifiers. Expression of the basic pigment cell defect appears variable, and genetic analysis is further complicated by the necessity for electron microscopic classification. The immune response essential to the completion of the amelanosis also appears to have a genetic component, as well as being affected by environmental factors that alter its activity (see Smyth, 1989 for review).

MELANIN AND SEX DETERMINATION

The value of sex determination for day-old chicks is well appreciated by both commercial poultry interests and poultry researchers. Separation of the sexes can be accomplished by either physical examination of the day-old chick's genitalia or by matings that result in sex-associated phenotypes. Both methods are widely used. However, the use of genetic markers for sex determination is less expensive, more convenient, and more accurate.

Two general approaches to using genetic markers in sex determination include the use of sex-linked crosses and within-line sex differences due to dosage effects of sex-linked barring (*B*) (see Silverudd, 1978 for review). The latter approach involves pure breeding lines and is generally referred to as autosexing. Jaap (1941) suggested that this term be used only for those lines where sex identification is definite, in contrast to some breeds like Barred Plymouth Rocks and Rhode Island Reds where pigmentation variations allow sexing with high but not complete accuracy (Jerome, 1939; Jaap, 1946). Silverudd (1978) refers to the latter type as semi-autosexing.

In general, sex-linked crosses have made a valuable contribution to the poultry industry, while autosexing stocks have received only limited attention. As pointed out by Silverudd (1974, 1978), a major problem for the autosexing lines is that they are not competitive with available commercial stocks. Designed for within-line sex determination, autosexing stocks have had limited use in mating schemes involving the production of crossbred and hybrid offspring. However, Silverudd (1978) has suggested a number of schemes whereby autosexing can be combined with sex-linked crosses for the efficient production of commercial poultry.

Sex-linked crosses. Two sex-linked mutations, barring (B) and silver (S) have been used extensively in sex-linked crosses for day-old sex identification. Two other traits, sex-linked albinism (s^{al}) and dermal shank pigmentation (id^{M}) have been proposed for this purpose, but as yet, have not been utilized on a practical basis.

Sex-linked barring. One of the first commercial sex-linked crosses to be utilized extensively involved the barring alleles, B and b^+. It was particularly popular for brown egg production during the 1930-1950s and is still used in some parts of the world. The so called 'black sex-link' pullet resulted from a mating of Barred Plymouth Rock females (B/W) with Rhode Island Red or New Hampshire males (b^+/b^+). All chicks were black due to the contribution of E by the dam, while the barred males (B/b^+) were identified by their white head spot and the females by its absence (b^+/W). Similar crosses in England also utilized Black Leghorn males as the nonbarred parent.

Variations exist in the coloration of the down and adult plumage of black sex-link females with some strains showing considerably more pheomelanin than others. Females ($E/e^{Wh}\ b^+/W$) with redder plumage generally have brownish-tinged black down, particularly on the head (Smyth, unpublished). Verdini (1980) found that the brown-face modification was accompanied by an increase in the spread of eumelanin into ventral regions of the down. The gene or genes responsible for the brownish pigmentation were not identified, although Db (dark brown), a logical candidate, was found not to be involved.

Sex-linked barring can also be utilized for chick sexing when on a blue-splashed white (Bl) extended black (E) background (Gawron and Smyth, 1980). The barring-induced white headspots are readily apparent on the $E/E\ Bl/Bl$ light blue-grey background. This combination results in essentially white plumage, and thus it has potential practical value for use in color sexing of meat stocks. The addition of silver (S) further eliminates the presence of pheomelanin.

Silver and gold. Sex-linked segregation of the silver (S) and gold (s^+) alleles is the basis for the most popular cross for the production of brown egg layers in the world today. Originally used on a columbian or black-tailed red background, differentiation of the silver male and gold female chicks was readily apparent as these phenotypes are expressed only in noneumelanic down. More recently, dominant white (I) has been added, since the pheomelanic down is still apparent on the I/i^+ female chicks. This has worked well for egg production stocks, where black-tailed reds were a usual part of the cross, but attempts to develop sexable crosses using meat stocks has been more difficult due to relatively low frequencies of genes that enhance the expression of silver and gold.

The key to silver-gold differentiation in the down was provided by Malone and Smyth (1979). They showed that silver and gold cannot be distinguished with accuracy on a wheaten background. In contrast, the addition of the columbian-like restrictor *Co* allows normal expression of *S* and s^+. In fact, the combination of dominant wheaten (e^{Wh}) and *Co* resulted in the clearest differentiation of *S* and s^+, either in the presence or absence of I/i^+. Other non-*E* alleles can also be used; however, selection may be desirable to enhance the expression of gold in the noneumelanized down. An exception to this involves the brown (e^b) allele, where silver-gold differentiation is difficult, particularly so in the presence of *I*.

Sex-linked albinism. The red eye color and short down associated with sex-linked albinism (s^{al}) was first suggested as a possible sexing agent by Hutt and Cole (1973). This approach would supply readily sexable chicks on all genetic backgrounds, including those resulting in white plumage. Its ultimate success will depend on whether its associated pleiotropies will prove to be a disadvantage commercially. The studies of Champion (1958), Hutt and Cole (1973), and Silversides and Crawford (1986, 1987) suggested that the sex-linked albino phenotype was normally productive and viable, while the sex-linked albino studied by Smyth et al. (1987) was associated with serious defects. Whether this discrepancy is due to different genetic backgrounds, environments, or alleles has yet to be determined.

Sex-linked dermal pigmentation. Although the dermal pigmentation associated with the id^+ or id^c alleles is not expressed in the down, a new allele (id^M) is apparent at hatching (Smyth and Boyle, unpublished). Therefore, id^M could be utilized for sex-linked crosses, particularly on an *E* background where its expression is enhanced, either in the presence or absence of recessive white (*c*). Its obvious disadvantage is the associated dark shanks and increased connective tissue pigmentation, particularly in the fascia of the skin in the abdominal area.

Saeki et al. (1954) reported that the offspring from a cross between Barred Plymouth Rocks and White Silkies could be sexed utilizing beak and comb color. Presumably, this relied on the sex-linked segregation of id^+ or id^c on a heterozygous fibromelanotic (Fm/fm^+) background. The use of shank color was less reliable, probably due to the presence of sex-linked barring and/or other hypostatic modifiers carried by the recessive white Silkies.

Autosexing. As pointed out by Silverudd (1974), sex-linked barring (*B*) could be called the autosexing gene, since it has been the key ingredient in all true autosexing stocks. Sex differentiation is accomplished through the well-established dosage effects of *B*. On appropriate genetic backgrounds, homozygous barring in males lightens down color appreciably more than does the hemizygous condition in females. This *B*-associated difference is apparent on a wide variety of phenotypes, but is most obvious on a striped down background.

The first autosexing breed was developed and described by Punnett and Pease (1930). It was derived from a cross between Barred Plymouth Rock and Gold Campine, an autosomal barred breed, followed by several backcrosses of barred cross-progeny to the Campine. Ultimately, birds that were essentially Campine, as well as pure for *B*, were developed. The line was then closed, with subsequent sex identification based on the lighter down color of males. Using the same general approach, the sex-linked barring gene was crossed into a number of pure varieties to create a host of autosexing breeds (see Hutt, 1949 and Silverudd, 1978 for review). The Englishmen, Punnett and/or Pease, were associated with the development of a number of these stocks, known collectively as the Cambridge breeds, while others were developed in western Europe and the United States. None appear to have made an important impact on the poultry industry.

Semi-autosexing. Since by definition (Silverudd, 1978) semi-autosexing does not result in complete accuracy, it has limited usefulness. For small groups of chicks and/or when vent sexing is not available, it is possible to obtain a high degree of accuracy by discarding the doubtful phenotypes. This would be useful when it is desirable to reduce the number of males to be reared, or to facilitate experimental designs where chick sex is important. Some examples of semi-autosexing within pure breeds are described below.

Barred Plymouth Rock. The sex-linked barring gene, again through its dosage effect on melanin pigmentation, results in sex differences. Females have smaller and less irregular white head spots, and generally darker down and shanks, than do males. In addition, the black pigment on the toes stops abruptly leaving the distal portion of each toe yellow. In contrast, males have larger blotchy irregular head spots and lack the sharp contrast between black and nonblack areas on the feet. Using these criteria, Jerome (1939) was able to sex Barred Plymouth Rock chicks with an approximate accuracy of 95 percent. However, as pointed out by Gerry and Mishler (1949), there are differences between strains and this may require some study and adjustment for a high degree of accuracy.

Rhode Island Red and New Hampshire. The presence of a white or light spot on the wing front of Rhode Island Red or New Hampshire day-old chicks has also been used for sex identification. Males have spots of varying sizes in the region of the wing web, while the wing color is uniform in females (Jaap, 1946). Jaap obtained sexing accuracies of 90 to 95 percent and 80 to 90 percent for Rhode Island Reds and New Hampshires, respectively. He also reported that accuracy could be improved by selecting breeders on the basis of their chick down color, for instance by selecting males with large white spots on the wings. The genetic and/or endocrine basis for the sex difference is not known, although it obviously does not involve the barring gene.

Buff Brahma. Down color in the Buff Brahma, a standard columbian-patterned breed, is characterized by dark dorsal pigmentation, the ventral down being cream-colored. Somes (1971) noted that the dorsal wing surfaces of females were smoky-grey, while on the male chick this area of the down was a clear creamy-yellow. Sexing accuracy using this white wing factor was approximately 93 percent. Although the factor segregated in outcross matings, its exact mode of inheritance was not determined. In addition, its relationship with the wing spot phenotype of Rhode Island Reds is not known, but the similar location of the light down in two different types of columbian-restricted chicks suggests that they may be related.

Light Brown Leghorn. MacArthur and MacIlraith (1946) reported that they could use down color to sex Light Brown Leghorns (e^+/e^+). Females had darker and more sharply defined dorsal back and head stripes. Based on this alone, they were able to sex with about 80 percent accuracy, and this was improved with the inclusion of other minor but consistent variations. They were not able to sex Dark Brown Leghorns, now known to carry the brown allele (e^b) at the E locus.

GENETICS OF PIGMENTATION IN SKIN, EYES AND OTHER NONFEATHER TISSUE

Pigmentation of nonfeather tissue involves the carotenoids, as well as the melanins. Xanthophyll, the most prevalent carotenoid, is found in many areas of the body, often sharing the same tissue with melanin. The combinations of the two lead to varying phenotypes, most obvious in yellow, white, green and blue shank colors. The deposition of both types of pigment in nonfeather tissue is also influenced by both specific individual genes and unidentified polygenic modifiers.

Carotenoid Pigmentation

The carotenoid pigments are responsible for the yellow coloration of the skin, shank, beak, body fat, and egg yolk. In contrast to its role in some other birds (for instance canaries, parakeets, flamingos), carotenoids play only a minor role in plumage color of chickens. They are apparent in nonmelanized chick down and developing feathers of white varieties. Presumably, they are also present in melanized down and feathers. An autosomal recessive mutation that eliminates carotenoid from the down has been described by Hutt (1951), who named it snow white and assigned the gene symbol, *sw*. Snow white had no apparent effect on melanic pigments, or on the presence of xanthophyll in the skin, shank or fat. According to Lamoreux and Hutt (1942) the carotenoid pigment in the down, unlike that found in fat and egg yolk, does not appear to be xanthophyll.

Carotenoids, mostly in the form of xanthophyll, are also present in the iris of the eye (Hollander and Owen,1939). In general, avian eye color is poorly understood and quite complicated (see Oliphant, 1988 for review). The carotenoids are associated with specific pigment cells termed chromatophores. They combine with the iridial vascularization to produce the common bay eye color of chickens. Carotenoids are not apparent in the eyes of newly hatched chicks and the bay eye is not fully expressed until sex hormonal increases occur prior to sexual maturity. Carotenoid levels decrease in females during extended periods of egg laying and are slow to return until postmolt feather growth is completed. This, plus the delayed appearance during early growth, led Kimball (1957, personal communication) to suggest that carotenoid deposition in the iris was delayed during periods of active feather growth.

Yellow skin color is a breed characteristic of Leghorn, Plymouth Rock, Cornish, Rhode Island Red, Brahma and many other breeds (see American Standard of Perfection, 1983 for review). In contrast, Orpington, Dorking, Sussex, Langshan, and Minorca, among other breeds have white skin. White skin in these breeds is associated with an autosomal dominant gene (W^+), although more recently a sex-linked recessive mutation (y) that converts yellow skin to white has been described (McGibbon, 1981).

Autosomal white skin. The major determiners of xanthophyll deposition in the skin are the autosomal white (W^+) and yellow (w) alleles, first described by Bateson in 1902, and subsequently confirmed by Dunn (1925) and others. White skin is considered to be wild-type, since W^+ is present in the red junglefowl. The white skin gene apparently acts to prevent the transfer of xanthophyll into the skin, beak, and shanks, since the iris of the eye, yolk and blood serum contain normal amounts.

The white and yellow skin phenotypes cannot be separated accurately until the chicks are 10-12 weeks of age. This is due to a delay in the deposition of xanthophyll after hatching in yellow-skinned chicks, while white-skinned chicks may show slight yellow coloration until 12 weeks of age. The situation is further complicated by the diet, whereby the carotenoid pigment content of such ingredients as yellow maize and alfalfa leaf meal enhance skin pigmentation, while rations replacing yellow maize with wheat, barley, milo or oats may result in very pale color in genetically yellow-skinned birds.

Variation in the intensity of yellow pigmentation of the shanks is also affected by sex and genetic factors. Breeds fed the same diets were found to differ significantly, while males had yellower shanks than females (Collins et al., 1955). Genetic effects were due to unidentified modifying genes that included a sex-linked component (Moyer and Collins, 1960; Zervas et al., 1962). The sex-linked effect could be due to the presence of the sex-linked barring gene in these stocks, since Jaap (1955) has shown that *B* lightens yellow shank color. Jaap's observations were on a silver columbian background, and he further indicated that the barring effect was dose dependent.

Sex-linked white skin. A sex-linked recessive mutation *y* also eliminates xanthophyllic pigment from the shanks and skin (McGibbon, 1981). Abdominal fat and yolks are yellow, but yolk color is lighter in eggs from y/W females than in eggs from either Y^+/W or birds homozygous for autosomal white skin (Lasher and Bitgood, 1982). Similarly, plasma levels of xanthophyll are reduced for sex-linked white-skinned birds. Sex-linked white skin differs from the autosomal type in that it can be identified accurately in newly hatched chicks. However, a role for *y* in practical sex-linked crosses appears unlikely, since white-skinned birds have significantly reduced growth rate and feed utilization (Patterson et al., 1983). It has been suggested that the mutant effect of *y* may involve the *de novo* pathway of phosphatidyl choline synthesis (Poernama et al., 1987).

Yellow head. An autosomal recessive mutation (*g*) associated with enhanced yellow coloration of the vascularized facial, comb and wattle tissue was described by Deakin and Robertson (1935). It is apparent at a few weeks of age, but is more striking prior to the onset of sexual maturity. After sexual maturity, male head furnishings approach the normal red color, while those of laying females become pale pink in color. The g/g genotype has no effect on the presence of the autosomal white skin gene (W^+). This defect is presumed to result from an interference with normal vascularization in the affected tissues.

Ear Lobe Color

Most breeds have red ear lobes due to the unmasked vascularization of the cutaneous tissue, although breeds of the Mediterranean Class, including Leghorn, Minorca, and Spanish, have enamel-white ear lobes. Interestingly, the red junglefowl shows a mixture of red and white, with red coloration in general predominating. The nature of the white pigment is neither carotenoid nor melanin, but a compound made up of purine bases (Louvier, 1934). In the White-faced Black Spanish breed, the white pigment covers the whole face, while in varieties such as the White Leghorn the spread of the pigment from the ear lobe into the facial skin, a common occurrence in older birds, is an undesirable characteristic.

The presence of the white purine pigment appears to be inherited as a polygenic trait (Warren, 1928). Presumably, its complete absence on one hand, and its extreme expression on the other, have resulted from the selection for modifiers that have altered the original partial occurrence in the wild-type progenitor.

Melanin Pigmentation Of Nonfeather Tissue

All melanocytes associated with pigmentation of nonfeather tissue, except for those of the retinal and iridial pigment epithelium, are thought to arise from the embryonic neural crest. After arrival, differentiation, and melanin synthesis, only those of the skin show further melanic activity. Nonfeather melanin is considered to be eumelanic, pheomelanins being found only in feather tissue.

Melanin may be found in the epidermis, dermis, eye, mesenteries, peritoneum, testes, around blood vessels, and in the connective tissue fascia closely adhering to the external abdominal muscles (see Lucas and Stettenheim, 1972 for review). Its presence or absence in individual tissues depends on the specific genotype, varying from complete absence in the tyrosinase negative autosomal albino (c^a) to the extreme melanization of the fibromelanotic phenotype of the Silky breed.

Dermal melanin. Although dermal melanocytes are rare in the body skin of most breeds, their presence in the dermis of the scaly shank is characteristic of several breeds. Blue-shanked breeds include the Hamburg, Polish, Campine, Fayoumi, Buff Catalana, and others. Green shanks are a breed characteristic of the Sicilian Buttercup. The blue shank coloration was shown by Barrows (1914) to be due to dermal melanin in the presence of melanin-free epidermis. The clear epidermal stratum corneum alters the appearance of the underlying black pigment to produce the blue coloration. The melanic basis for green shanks is identical, the blue and green color difference being due to the absence or presence of yellow carotenoid pigment, respectively (Dunn, 1925).

Dermal melanization is controlled by alleles located on the Z chromosome (Punnett, 1923; Dunn, 1925). Wild-type dermal melanin was found to be recessive to its inhibitor, the allelic symbols originally being *d* and *D*, respectively. These were later changed to id^+ (dermal melanin) and *Id* (inhibitor of dermal melanin), thereby relinquishing the *D* symbol to represent duplex comb. Dominance of *Id* is incomplete, since heterozygous males show faint but distinct dermal shank melanin pigmentation.

Early investigators of the effects of the id^+ allele (see Hutt, 1949 for review) reported that it was not expressed until approximately three months of age and that it was independent of genes controlling epidermal and feather pigmentation. Dermal shank pigment is not eliminated by either dominant (*I*) or recessive (*c*) white but both dilute it (Knox, 1935). Other inhibitors of id^+ include barring, mottling, both kinds of wheaten (Nordskog, 1970; Smyth, unpublished), and the columbian-like dilute gene *Di* (Brumbaugh and Hollander, 1966).

More recent work indicates that the *Id* locus has multiple alleles. Two new alleles, id^c and id^a, were proposed by McGibbon in 1974 and 1978, respectively, while the author is currently investigating an apparent fifth allele, id^M (Smyth, unpublished). The descriptions of these are as follows:

id^c. The id^c mutation occurred in the Cornell Randombred White Leghorn line (hence the *c* superscript) and differed from id^+ in that plumage and beak pigment is present in the dominant white (*I/I*) mutants (McGibbon, 1974). In addition, McGibbon (1979) reported that green-shanked id^c females were significantly more susceptible to tumors (mainly hemangiomas) than were their sisters with yellow shanks (*Id*).

id^a. McGibbon (1978) studied shank color in the Ancona breed and attributed the presence of small green spots to be due to the effects of a new *Id* allele, id^a, on dermal melanin. However, the data presented do not convincingly indicate a separate allele. Interactions between the mottling gene of the Ancona and other modifiers could also explain these data.

id^M. The Massachusetts mutant appeared in a synthetic line derived from a cross between Black Langshan and a dominant white meat line. It differs from the above in that it is expressed in the shanks at day-old in black (*E*), birchen (E^R), wild-type (e^+) and recessive white (*c*) chicks. It produces the darkest shank color when combined with *E*. In the presence of *c/c E/-*, id^M shank color is somewhat diluted, but it is still dark blue. The id^M allele is not expressed in the shanks of dominant white (*I*) chicks, but the combination of *I* and *E* results in a pale blue or green color by 10-12 weeks of age.

In light of the multiple allelic nature of the *Id* locus, it is interesting to speculate on the possibility that earlier workers may have studied several alleles under the guise of id^+. For example, Punnett (1923) noted that the sex-linked shank pigment gene was expressed at day-old in some matings, but not in others. Could this have been id^+ and id^M or similar alleles? In addition, Jaap (1955) found that sex-linked barring resulted in dermal shank pigment on a silver columbian background in the absence of id^+. Therefore, barring can mimic an id^+ effect on certain genetic backgrounds.

Epidermal melanin. In general, the epidermis of the skin contains very few pigmented melanocytes, the eyelid of many genotypes and the facial epidermis of certain dark gypsy or mulberry-faced varieties being exceptions.

The major genes responsible for epidermal melanization in the chicken are the black extension factor, *E*, and its less effective birchen allele, E^R (Moore et al., 1972). The effects of *E* are most apparent on the shank epidermis, where in the absence of certain eumelanin inhibiting genes the shanks are almost black. Black shanks that are due to *E*/*E* also have some dermal pigmentation even in the presence of *Id* (Barrows, 1914). The effects of the birchen (E^R) allele are more readily inhibited. The major eumelanin shank pigment inhibitor is dominant white (*I*), which also removes black pigment from the feathers. The secondary pattern genes sex-linked barring (*B*) and mottling (*mo*) also inhibit shank melanization. The effect of *B* is dose dependent, so males (*B*/*B*) have lighter shanks than do hemizygous females. Motting is expressed in the shanks as small scattered black spots on a noneumelanized background.

Epidermal pigmentation of the skin of the face, comb and wattles is peculiar to the gypsy or mulberry face phenotype. Gypsy face is a breed characteristic of Sumatras, Birchen and Brown Red Modern Games, and to a lesser degree of Sebrights. Evidence that epidermal melanin is involved is based on fancier reports that sunlight causes darkening of the facial pigment. In contrast, Auclair (1987) was not able to alter facial pigmentation with ultraviolet light in birds with dermal pigment only, as in either those carrying the dermal melanin factor id^+, or those having the fibromelanosis phenotype. Based on crosses between Silver Sebrights and Golden Pencilled Hamburgs, Punnett (1923) concluded that the dark facial pigment behaved as a dominant gene and was closely related to dark down color. He also reported the phenotype as being present in Spangled Hamburgs and Campines. Since all of the gypsy-faced stocks must have carried either black extension (*E*) or birchen (E^R), it would appear that either one of these, or some similar allele, is involved. However, most black and birchen phenotypes have bright red faces apparently devoid of melanin, including those that carry the dermal melanin factor (id^+). It is not possible to say at this time whether the gypsy face phenotype is due to a different *E* allele or to a dominant modifier of *E* or E^R.

Genetic basis for shank color phenotypes. The phenotypic expression of shank and foot color is dependent on the cumulative and interactive effects of several major genes, plus unidentified modifiers that enhance or restrict eumelanin (see Knox, 1935; Sturkie et al., 1937; Moore et al., 1972 for review). The effects of the *Id* and id^+ alleles are confined to the dermis, while *E* is primarily associated with the epidermis, but does add varying amounts of dermal pigment. Dominant white (*I*) is epistatic to epidermal melanin, and it dilutes but does not eliminate dermal pigment. Recessive white acts similarly. Sex-linked barring and mottling are major inhibitors of shank eumelanin. In addition, the absence or presence of yellow carotenoid pigment, due to the W^+ and *w* alleles, respectively, interacts with the melanin status of the dermis resulting in either blue or green shank color.

It is surprising in light of the number of genetic analyses of shank color inheritance that there is still considerable unexplained variation. Some of the unexpected segregations observed by Knox (1935) and Sturkie et al. (1937) could be accounted for by some of the more recently identified alleles at the *E* and *Id* loci. Moore et al. (1972) demonstrated that the extended black (E) allele is associated with much darker shanks than is the birchen (E^R) allele. Birchen can be, however, present in birds with self-black plumage if the necessary eumelanin enhancers are also present. This could account for self-black birds with little or no shank melanin. The occurrence of light greenish pigment in the shanks, or portions of the shanks, in the apparent absence of id^+ is still puzzling. Sex-linked barring eliminates the dermal pigment in both recessive and dominant white birds (Jaap, 1943), but enhances it in the presence of the columbian phenotype (Jaap, 1955). The latter also reported that barring reduces the intensity of yellow shank coloration.

A summary of the interactions involving the major pigmenting genes associated with shank color is presented in Table 5.6.

Fibromelanosis. The hypermelanic pigmentation of the Silky breed attracted early scientific analysis (see Hutt, 1949 for review). Since the characteristic dark bluish skin was due to dermal and not epidermal pigment, it was considered to represent enhanced mesodermal pigmentation. However, since the melanocytes of both the epidermis and dermis arise from the ectodermal neural crest, Hutt (1949) suggested the name fibromelanosis, to indicate its association with connective tissue (Kuklenski, 1915; White and Eastlick, 1953; Stolle, 1968).

Fibromelanosis is characterized by extensive pigmentation of the dermis, particularly in association with the loose connective tissue of the 'deep dermis', as well as of the parietal and visceral peritoneum, the periosteum, and the perichondrium. In addition, numerous melanocytes are found around blood vessels and nerves, and in association with the

Table 5.6. Major genotypes associated with shank and foot color inheritance.

Pigment status				
Carotenoid	Dermal melanin	Epidermal melanin	Genotype	Phenotype
W^+	Id	E	$W^+/W^+\ Id/Id\ E/E$	Near-black with white soles
		e^+	$W^+/W^+\ Id/Id\ e^+/e^+$	White shanks and feet
	id^+	E	$W^+/W^+\ id^+/id^+\ E/E$	Black shanks with white soles
		e^+	$W^+/W^+\ id^+/id^+\ e^+/e^+$	Blue shanks with white soles
w	Id	E	$w/w\ Id/Id\ E/E$	Near-black with yellow soles
		e^+	$w/w\ Id/Id\ e^+/e^+$	Yellow shanks and feet
	id^+	E	$w/w\ id^+/id^+\ E/E$	Black shanks with yellow soles
		e^+	$w/w\ id^+/id^+\ e^+/e^+$	Green shanks with yellow soles

connective tissue stroma of the viscera. The appearance of fibromelanotic pigment in the embryo occurs much later (13-13.5 d) than in normally pigmented tissue (White and Eastlick, 1953). Prior to eight days of incubation, the numbers of connective tissue-associated melanocytes are approximately the same for Silkies and Brown Leghorns (Stolle, 1968). At this point, the Silky fibromelanotic melanocyte numbers increase sharply, while Brown Leghorn pigment cells die and are phagocytized. Based on cell culture studies, Wendt (1958) concluded that Silky-conditioned media contained a pigment cell activator that was missing in the Brown Leghorn media. However, Stolle (1968) made reciprocal chorioallantoic grafts between Silky and Brown Leghorn embryos and found the graft tissue to pigment independently of the host environment.

Several early investigators concluded that the inheritance of fibromelanosis involved a dominant sex-linked inhibitor. Dunn and Jull (1927) concluded that this was the dermal melanin inhibitor *Id*, or a closely Z-linked gene. Stolle (1968) reported that fibromelanosis is due to sex-linked id^+ in concert with a dominant enhancer, earlier assigned the gene symbol *Fm* by Hutt (1949). According to Stolle, combinations of these genes resulted in the following grades of connective tissue pigmentation:

$Fm/-$ id^+/W (or id^+/id^+) - darkly pigmented
$Fm/-$ Id/W (or $Id/-$) - faintly pigmented
fm^+/fm^+ id^+/W (or id^+/id^+) - unpigmented
fm^+/fm^+ Id/W (or $Id/-$) - unpigmented

Based on shank color alone, Auclair (1987) did not observe pigment in $Fm/-$ Id/W females. Earlier workers reported considerable variation in the degree of fibromelanotic pigmentation, suggesting that a number of other factors can modify its expression. Some of these may also be related to plumage color, although fibromelanotic Silkies exist with white, black, blue and partridge patterns.

Pigmented abdominal skin fascia. The presence of eumelanin in the abdominal skin fascia of the dressed carcass is unattractive to the consumer. Located in fascia closely adhering to the external abdominal muscles, it varies from a few spots to a complete covering of the posterior half of the two muscles (Jaap, 1958). Color intensity varies from light grey to black. It occurs in both sexes, but the incidence is significantly higher in females (Jaap, 1958; Huntsman et al., 1959; Kuit, 1967). Jaap (1958) also noted that it was often associated with the non-id^+ dermal pigmentation described previously.

The genetic bases for abdominal fascia eumelanization involve a series of complicated interactions between known and unknown genes, as well as incomplete dominance between some of the alleles. Dominant white (*I*) inhibits the abdominal pigment, particularly in the presence of extended black (*E*), sex-linked barring (*B*), and possibly silver (*S*) (Jaap, 1958). Similar results were reported by Huntsman et al. (1960), although they found the combination of *I* and *B* to have no more inhibitory effect than either *I* or *B* singly. Extended black (*E*) birds were freer of abdominal pigmentation than columbian-restricted birds (Huntsman et al., 1959), while barring increases the incidence in columbians, as does its allele B^{Sd} according to Kuit (1967). Wilkins (1975) observed that e^+ (wild-type) and e^b (brown) are associated with a higher incidence of abdominal pigment than *E*, while no consistent relationship was associated with the *Co* (columbian restriction) gene. This probably explains the previous observations on *E* and columbian phenotypes, and suggests the latter were e^b or e^+ at the *E* locus, since Wilkins also showed that the wheaten allele (e^{Wh}) was a strong inhibitor of the fascia pigment. Recessive white (*c*) appears to enhance abdominal melanin, particularly for chicks with smoky down color (Jaap, 1958). Although it is probably not involved in the earlier studies using commercially acceptable stocks, the dermal melanin gene (*id*$^+$) causes a marked increase in abdominal pigment (Kuit, 1967; Wilkins, 1975).

Based on published data the deposition of abdominal fascial pigment appears to be a sex-linked trait in some cases and a sex-limited trait in others. Sex-linked effects probably involve sex-linked barring (*B*). The sex-limited aspects could be explained as being due to a sex-linked gene with inhibiting effects that had no allelic counterpart in the populations studied (Huntsman et al., 1959), particularly since no direct effect of sex hormone treatments on the expression of abdominal pigment was found by Jaap (1958).

In summary, the above data suggest that the most effective genotype for the prevention of eumelanization of the fascia of abdominal skin is the homozygous combination of dominant white (*I*), nonrecessive white (C^+), extension of black (*E*), sex-linked barring (*B*), silver (*S*) and inhibition of dermal melanin (*Id*).

Eye Pigmentation

There is a surprising lack of information on the subject of the inheritance of eye color. In fact, in their respective poultry genetics books, Jull (1952) limited comment on the subject to two short paragraphs, while Hutt (1949) chose not to consider eye color under a separate heading. Since then, very little new information has been published. In part, this is due to the complexities of the trait, and to the fact that most available information is related to macroscopic eye color rather than to pigmentation of specific ocular tissues. The situation is further complicated by age-related changes. All chicks except those in the albinotic group (c^{re}, c^a, s^{al} and *pk*) have dark eyes at hatching. Final eye color is not achieved in most cases until the attainment of sexual maturity, when the carotenoid and melanin pigments are fully expressed. After the onset of egg production, female eye color may change again because of the reduction or absence of carotenoids in association with egg yolk production.

The various structures of the eye are pigmented to varying extents depending on the specific genotype. Two pigment cell populations varying in their embryonic origin are involved. The melanocytes of the retinal, ciliary, and iridial pigment epithelium, and probably the pectin, are derived from the optic cup (Hodges, 1974). Those populating the uveal tract, including the choroid, the bulk of the ciliary body, and the anterior surface of the iris, originate in the neural crest. The iris, which plays a major role in eye color, can have pigment on both the anterior and posterior surfaces, as well as melanocytes within the interior connective tissue stroma, particularly surrounding blood vessels.

The wild-type eye, as characterized by the Light Brown Leghorn, has a heavily melanized retinal pigment epithelium and pectin, along with a double layer of pigmented epithelium on the posterior surface of the iris. Only a few scattered melanocytes are found on the anterior surface, while the rest of the uveal tract is heavily pigmented. The interior stroma of the iris is devoid of pigment. The addition of carotenoids in the Brown Leghorn produces the bay eye phenotype. The bay color is the combined effect of the iridial blood vessels, the carotenoids, and the iridial pigment epithelium on the posterior surface of the iris. Brown-eyed phenotypes show an increase in melanized pigment cells on the anterior surface, which may appear in a solid or flecked pattern. The darkest eye color is associated with the fibromelanotic phenotype, characterized by heavy deposits of melanin throughout the eye, including the sclera. Stromal pigmentation, however, is still primarily perivascular. The author is aware of little information about the basis for pearl eye, characteristic of the Cornish and Aseel breeds. It would appear to have the melanin distribution of the bay eye, in the absence of detectable carotenoid pigment.

Early studies involved crosses between brown-eyed breeds with black plumage and bay-eyed stocks with restricted black plumage. There was a general awareness of a relationship between eye color and feather color, as well as one between melanized shanks and darker eye color. Although this indicated that genes associated with plumage and skin pigmentation also affect eye color, there were enough exceptions to confuse the situation and discourage further investigation.

Most early investigators agreed that a dominant sex-linked factor inhibits brown eye color (Punnett, 1923; MacArthur, 1933; and Kimball, from an unpublished manuscript in the author's possession, circa late 1940s). However, it is not known whether or not they were working with the same loci and/or alleles. Punnett's inhibitor appears to have been the dermal melanin inhibitor (*Id*). The id^+ or id^M alleles enhanced both dermal shank and eye pigmentation. These results agree with observations from the author's studies, involving id^+ and id^M, that eye color is closely related to shank color. The hypothesized id^M allele not only causes dark brown eyes in the presence of black (*E*) plumage, but its darkening effect on the eyes of wild-type (e^+) is also readily apparent.

In contrast, MacArthur (1933) observed light and brown-eyed phenotypes in the presence of either light or dark skin and all shank colors. Similarly, two pure varieties of the Japanese bantam (the Black and the Grey) feature brown eyes in the absence of dermal melanin. MacArthur (1933) attributed the sex-linked segregation observed in his study to two previously unidentified alleles, *Br*, an inhibitor of brown eye, and *br*, which allows melanin deposition in the eye. He further showed the *Br* locus to be separated from the *Id* locus by approximately 22.5 crossover units. The *Br* alleles appear to affect only eye pigmentation. Kimball (unpublished) also made a series of eye color matings and attributed certain sex-linked effects to *Br* rather than to *Id*. Although it is not known which locus was involved, both Bateson and Punnett (1911) and Dunn and Jull (1927) reported that a sex-linked dominant gene was capable of inhibiting the extremely melanized eyes of fibromelanotic chickens.

Eye color can also be modified by other genes known to be associated with plumage color. The black plumage alleles *E* and E^R result in a brownish eye in the absence of specific inhibitors. The *E* allele is associated with a darker eye than E^R, and in concert with the dermal shank alleles id^+ and id^M, it results in a dark brown eye. Sex-linked barring is another inhibitor of eye melanization (Kimball, unpublished; Stradtner et al., 1988), and undoubtedly other eumelanin inhibitors like wheaten (e^{Wh}) also exert an effect. Recessive white (*c*) appears to have little effect on eye color, while dominant white (*I*) is epistatic in the author's experience to brown eye color, possibly due to its effects on extended black (*E*).

The genetic basis for pearl eye has not been determined, but it appeared to be recessive to brown eye in a cross between pearl-eyed Malays and bay-eyed Buff Orpingtons (Bond, 1919). That pearl is not due to the white skin gene (*W*) is shown by the fact that pearl-eyed Cornish have yellow skin.

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Chapter 6. MUTATIONS AND MAJOR VARIANTS OF PLUMAGE AND SKIN IN CHICKENS

Ralph G. Somes, Jr.

In this chapter all of the inherited modifications of chicken plumage and skin, excluding differences in color and pattern, are described. Modifications of the plumage are basically of two types, those which affect the feather tracts by reducing their size or by completely inhibiting their development, and those which modify the feathers by acting on their follicles. All of them are listed in Table 6.1. Gene action includes prevention of feather development, changes in feather structure, and changes in feather growth rates. Not as many structural variations of the skin and related structures have been described in the literature. They will be described by grouping them according to the part most affected — comb, uropygial gland, spur, and skin coat. They are listed in Table 6.2. A detailed account of skin and feather development, structure, and growth is beyond the scope of this book. For information on these subjects the two-volume reference by Lucas and Stettenheim (1972) is highly recommended.

PLUMAGE VARIANTS

Normal Feathers

In normal chickens, the main feathers are located in tracts or *pterylae*, of which there are ten. The spaces between the tracts are called the *apteria* and they usually contain scattered down feathers and semiplumes. Chicken shanks and feet are usually not feathered.

There are two general kinds of feathers. The *neossoptiles* are the down feathers of newly hatched chicks. They consist of a large number of barbs attached to a common base giving the fluffy appearance to the chick. The *teleoptiles* include adult feathers and juvenile feathers that replace the chick down. These feathers are basically of three types: the *plumules*, which are downy feathers of the abdominal; the *filoplumes*, which are long hair-like feathers found in the apteria; and the *pennae* or *contour* feathers, which include all those with a well developed central shaft. The *remiges* (primaries and secondaries of the wing) and *rectrices* (tail feathers) are also contour feathers.

The main features of contour feathers are the *shaft*, *vane*, and usually an *afterfeather* on the undersurface at the proximal end of the vane. The portion of the shaft supporting the vane is called the *rachis* and it is essentially the solid portion of the shaft above the skin. The lower portion of the shaft is called the *calamus* and it is tubular. The junction between the rachis and the calamus is marked by a small opening on the ventral surface, the *superior umbilicus*. At the lower end of the calamus is another opening, the *inferior umbilicus*. The vane is made up of the web of the feather on either side of the rachis. Many *barbs* form the web and they all lie in the same plane. Each barb carries *distal barbules* directed toward the feather tip and *proximal barbules* directed toward the base. Barbules in turn carry small projections known as *barbicels*. As the barbules on adjacent barbs obliquely cross each other, their barbicels interlock forming the compact web of the feather.

Table 6.1. Listing of plumage variants in chickens.

Trait and gene symbol		Trait and gene symbol	
Variations in feather distribution			
Naked neck	*Na*, *na*$^+$	Congenital baldness	*ba*, *Ba*$^+$
Apterylosis	*Ap*, *ap*$^+$	Edema	*ed-1*, *Ed-1*$^+$; *ed-2*, *Ed-2*$^+$
Scaleless	*sc*, *Sc*$^+$	Ptilopody	*Pti-1*, *pti-1*$^+$; *Pti-2*, *pti-2*$^+$; *pti-3*, *Pti-3*$^+$
Ottawa naked	*nk*, *Nk*$^+$	Stubs	*Ht*, *ht*$^+$; *sb-1*, *Sb-1*$^+$; *sb-2*, *Sb-2*$^+$
Variations in feather length			
Crest	*Cr*, *cr*$^+$	Eight tail feathers	*ext*, *Ext*$^+$
Muffs and beard	*Mb*, *mb*$^+$	Surplus primaries	*Sf1*, *sf1*$^+$;*Sf2*, *sf2*$^+$
Vulture hocks	*v*, *V*$^+$	Long filoplumes	*Lf*, *lf*$^+$
Long tail	*Gt*, *gt*$^+$; *mt*, *Mt*$^+$		
Variations in feather structure			
Feather texture	*Ha*, *ha*$^+$	Tail feather hypoplasia	*Hy*, *hy*$^+$
Frizzling	*F*, *f*$^+$; *mf*, *Mf*$^+$	Matted down	*
Silkiness	*h*, *H*$^+$	Ragged wing	*rw*, *Rw*$^+$
Naked	*n*, *N*$^+$	Porcupine	*pc*, *Pc*$^+$
Wing patch		Ropy	*ropy*, *Ropy*$^+$
Abnormal feathering	*af*, *Af*$^+$	Stringy	*st*, *St*$^+$
Alopecia	*	Stringy-2	*st-2*, *St-2*$^+$
Dysplastic remiges	*dr*, *Dr*$^+$	Sunsuit	*sn*, *Sn*$^+$
Flightless	*Fl*, *fl*$^+$	Wiry	*wi*, *Wi*$^+$
Fray	*fr*, *Fr*$^+$	Woolly	*wp*, *Wp*$^+$
		Henny feathering	*Hf*, *hf*$^+$
Variations in feather growth rate			
Slow feathering	K^n, K^s, K, k^+	Modified slow feathering	*
Retarded-tardy	t^s, t, T^+		

* Probably polygenic.

Variations In Feather Distribution

Naked neck (*Na*). Naked neck chickens, often referred to as Turkens, Transylvania Naked Necks, Bare Necks, Hackleless, and Rubber Necks, are characterized by the naked neck trait. This unusual trait was first studied by Davenport (1914) who found it to be caused by a single autosomal dominant gene, which Warren (1933a) later confirmed. The gene symbol *Na* was assigned by Hertwig (1933). The *Na* gene is actually incompletely dominant with *Na*/*na*$^+$ birds showing an isolated tuft of feathers on the ventral side of the neck above the crop, while *Na*/*Na* birds either lack this tuft or it is reduced to just a few pinfeathers or small feathers (Crawford, 1976). Scott and Crawford (1977) demonstrated that the presence or absence of the tuft could be used to identify the two genotypes accurately at hatching.

Greenwood (1927) made a detailed description of the modified feather distribution resulting from the *Na* gene. Normally the apteria carry scattered down and semiplume feathers, but the apteria of naked neck birds contain no feathers. The feather tracts themselves are also either absent or reduced in area so that birds have greatly reduced feather cover. Classen and Smyth (1977) verified this by showing a 30 percent increase in the lateral pelvic apteria width of *Na/Na* chicks compared to *Na/na*$^+$ chicks. The reduction was less in heterozygotes than in homozygotes, being 27 and 22 percent for *Na/na*$^+$ females and males respectively and 41 and 33 percent for *Na/Na* females and males (Bordas et al., 1978). The capital tract of the head is absent except for around the comb. The dorsal and ventral cervical tracts are absent. The dorsopelvic, dorsal caudal and pectoral tracts are all markedly reduced in area. The resulting bare skin becomes reddish, particularly in males as they approach sexual maturity.

Refer to Chapter 20 for a discussion on abnormal transmission of the *Na* gene and on the superior heat tolerance that is associated with it.

Apterylosis (autosomal nakedness) (*Ap*). Sturkie (1942) studied an autosomal naked condition in a Rhode Island Red flock, and Hutt (1949) later assigned the name apterylosis and the gene symbol *Ap*. This nakedness was somewhat like the naked neck trait in that it was due to reduced or absent pterylae, but it was more extreme than naked neck. Expression of the condition varied from almost normal to almost completely naked. Where follicles were present, feathers developed normally, but down and semiplumes were always absent in all apteria. The tracts most affected were the anterior and posterior spinal, humeral, and femoral. In extreme cases the ventral tracts were small or absent. Head feathering was usually normal with areas over the eyes being the only denuded area. The wing and leg tracts were always normal.

Genetic crosses showed this type of nakedness to be due to a single dominant autosomal gene with no apparent difference between homozygous and heterozygous individuals. Random selection over eight generations resulted in the accumulation of genetic modifiers against its more extreme expression (Sturkie, 1950).

The *Ap* gene had no adverse effect on hatchability, but it was lethal to about 58 percent of the mutant chicks by 15 days of age due to reduction in their feed intake. When fed wet mash, feed intake increased and mortality decreased. However, this was only temporary, since mortality again increased with the discontinuing of wet mash feedings (Sturkie, 1942). The lethality associated with this trait was not due to a lack of feathers. It was associated with reduced feed intake, not as a primary cause, but as a consequence of some more basic physiological disorder. At time of hatch, body size was comparable to normal chicks, but it was only 80-90 percent that of normal birds later. At maturity, the naked birds were about 75 percent of the weight of normals.

Scaleless (*sc*). A naked condition in which feather papillae, scales, foot pads, and spurs do not develop was reported by Abbott and Asmundson (1957) in a New Hampshire flock (Figure 6.1). This extreme condition has been named scaleless. Scaleless chicks were nearly completely lacking in down feathers at hatch, although by selection it was possible to increase the amount of down and subsequent feather cover, and high and low feather lines have been developed (Abbott and Asmundson, 1962). The epidermis was transparent, waxy, and smooth. Shanks were devoid of scales, as were the foot pads, and both were smooth in texture. Hatchability was not affected by this condition, but special brooding conditions were needed to avoid posthatch mortality. The few feathers that did develop on adults were primitive in structure. Males did not develop spurs although other ectodermal derivatives such as nails, beak, comb, and wattles were normal. As scaleless birds matured, their skin became reddish, this being particularly noticeable with males.

Genetic studies revealed that scaleless was inherited as a single autosomal recessive gene to which the symbol *sc* has been assigned (Abbott and Asmundson, 1957).

Many studies have been made involving this gene. Sengel and Abbott (1963) showed

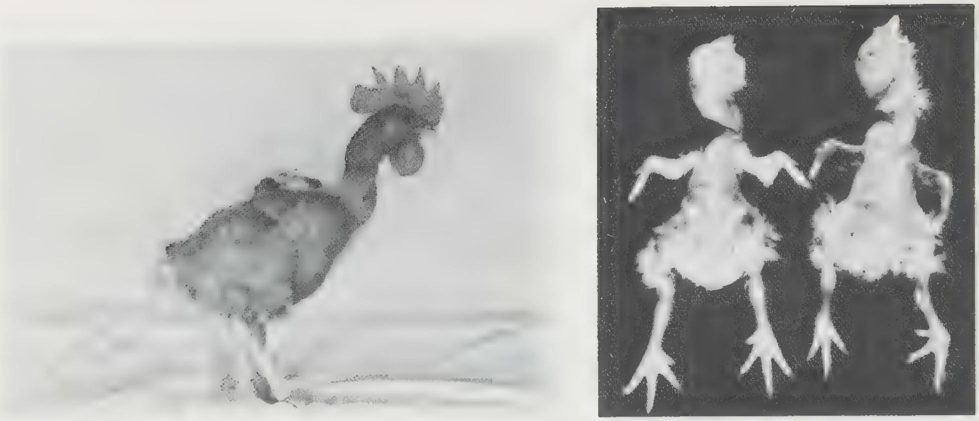

Figure 6.1. The scaleless (*sc*) mutant. Left — adult male (author's photo). Right — day-old chicks (from Abbott and Asmundson, J. Hered. 48:65, 1957).

that the scaleless dermis is able to function normally and that the epidermis alone is responsible for the condition. Morphogenic studies of both the dorsal skin (Goetinck and Sekellick, 1970, 1972) and the shank skin (Sawyer and Abbott, 1972) have been made, and Brotman (1976, 1977a, 1977b, 1977c) has made similar studies using the selected high and low feather lines. The effect of *sc* on the development of the conjunctival papillae and scleral ossicles in the eye has also been studied (Palmoski and Goetinck, 1970). Research has been conducted on nutrition (Abbott et al., 1962), heat production (Abbott and Asmundson, 1958; Wekstein and Zolman, 1970), heart rate (Cain and Abbott, 1971), organoleptic qualities (Bigbee and Rubin, 1973; Somes and Wiedenhefft, 1982), carcass composition (Somes and Johnson, 1982), and growth in various environments (Johnson and Somes, 1978; Somes and Johnson, 1982).

Ottawa naked (*nk*). The Ottawa naked mutant first observed in 1956 is the newest naked mutation to be reported in the domestic fowl. Affected chicks are almost completely devoid of down feathers in extreme cases or they may have just a few bare patches. Naked areas are devoid of feather follicles so that as adults they are as naked as they were as chicks. The third and fourth toes are characteristically fused for most or part of their length. The chicks can be raised to adulthood by using special care and their reproductive rate is normal when artificial insemination is used (Crawford et al., 1982). Other associated defects, besides absence of feathers and syndactyly, are spinal cord and kidney defects, embryonic edema, and scale development defects (Fulton et al., 1987). Embryonic mortality of homozygotes was found to be very high, due presumably to the kidney and spinal cord defects.

Crawford et al. (1982) reported that this condition was due to an autosomal recessive gene with complete penetrance and variable expressivity. Etches and Hawes (1973) gave it the gene symbol *nk*. Because all affected structures either differentiate from or depend on the mesoderm, it has been suggested that the site of gene action is within this germ layer (Fulton et al., 1987).

Congenital baldness (*ba*). Sometimes among newly hatched chicks there will appear individuals with small bald areas on the top of their heads. Hutt (1932) referred to these areas as supraorbital featherless areas, whereas in later studies (Jaap, 1937; Sturkie, 1941; Somes, 1970a, 1989) the condition was called congenital baldness. This condition is quite common. It has been reported many times in many different genetic stocks (Hutt, 1932; Jaap, 1937; Somes, 1970a, 1989; Sturkie, 1941). The irregular featherless areas are either above the eyes or posterior to the comb. Feather follicles are absent from these areas and affected areas vary in size, from very small with just one or two follicles missing, to areas as large as ten mm^2. Distribution of bald areas varies widely among the three affected areas and to a lesser degree between sexes. Jaap (1937) never saw bald areas in all three locations on the same chick, but Somes (1989) observed three percent of 546 bald chicks to be of this type. Sturkie (1941) studied the embryological and physiological basis for bald area formation. He concluded that an inherent defect of the epidermis caused a separation of the dermis and epidermis in the affected area, which then allowed for a fluid-filled bleb to form. Blebs usually formed at eight days of development, reached maximum size by 11 days, and regressed after absorption of the fluid by 15-16 days. During this critical period, feather papilla formation was interfered with leaving a smooth bare-skin bald area.

Hutt (1932) suggested that the trait was inherited as a simple recessive. Jaap (1937) could not get the trait to breed true and concluded that it was either multifactoral or that some condition in the embryo's environment was responsible for inhibiting full expression of baldness. He also showed that there was no relationship between location of the parent's bald areas and those of their offspring. Sturkie (1941) assumed that the trait was due to a recessive gene with incomplete penetrance. Somes (1989) made extensive crosses which led him to suggest that congenital baldness was inherited as a sex-linked recessive with a linkage relationship to the s^{al} locus of about eight cross-over units. These data, however, also suggested that some heterozygous males showed the trait. Thus, either the trait is not completely recessive or several genes are involved with one of them being sex-linked. The gene symbol *ba* was assigned for the sex-linked gene involved with this trait (Somes, 1989).

Edema (thigh patch) (*ed-1,ed-2*). Savage et al. (1986) described a condition which resulted in featherless areas on either one or both thighs. Fluid-filled sacs detected as early as 12 days of incubation prevented feather follicle development similar to that for the congenital baldness trait. Sacs ranged in size from 0.5-4.0 cm diameter in 16-day-old embryos. Although chicks hatched and could be raised to maturity, hatchability was greatly reduced by this disorder. Pedigree matings led to the suggestion that the mode of inheritance for this trait was that of two autosomal recessive genes at two separate loci. It is proposed here that the gene symbols *ed-1* and *ed-2* be assigned to these loci.

Feathers on the feet or ptilopody (*Pti-1, Pti-2, pti-3*). Feathers on the feet are referred to by several names: shank feathering, feather-legged, feathered feet, and ptilopody. They are characteristic of several breeds. The phenotypes associated with this trait differ greatly. The Breda, Langshan, and Faverolle breeds show the least expression with only the outer surface of the tarsometatarsus and the outer toe being feathered. The Brahma and Silky breeds also have feathering only on the outer surface of the shanks, but on both the outer and middle toes. The Belgian d'Uccle Bantam, Booted Bantam, Cochin, and Sultan have extensive feathering on both the outer surface and front of the shanks and on both the middle and outer toes. In the Pavloff breed shank feathering is quite different; although the feathering is not particularly heavy, it exists on the inner surface of the shanks as well as the outer and front, and it is present on all four toes.

Many studies have been made in an attempt to determine the genetic basis for feathers on the shanks and toes. Hurst (1905) was one of the earliest investigators of this trait; using the Cochin breed, his results indicated partial dominance, but it was later pointed out by Punnett and Bailey (1918) that these results could be explained on the basis of two loci with one member of either pair of genes being sufficient to produce feathered shanks. Davenport (1906, 1909) reported extensively on this trait; he concluded that feathering was a recessively inherited trait closely associated with a feathering inhibitor. Punnett and Bailey (1918), however, suggested that Davenport's work with Brahmas and Cochins could also be explained by the action of two dominant duplicate factors. Punnett and Bailey (1918) using the Croad Langshans concluded that shank feathering was due to one pair of dominant genes. Serebrovsky (1926) made probably the most detailed of the feathered-shank investigations. He showed that Pavloff feathering was due to a single recessive gene, although about 40 percent of his heterozygotes showed a 'weak' type of shank feathering. He proposed two dominant genes to explain his work with the Brahma breed. Dunn and Jull (1927) studying Silkies concluded that two dominant genes were involved in two of their crosses but only one pair of genes appeared to be involved in a third. Lambert and Knox (1929) working with Langshans obtained results that suggested that some of their parental Langshans carried two pair of genes for leg feathering, some were apparently heterozygous at one locus, and some might have had only one pair of genes. Warren (1933a) also reported results from Brahma and Silky crosses; his backcross data indicated that both breeds carried a dominant gene for leg feathering.

Somes (unpublished), using Langshan, Brahma, Cochin, and Sultan crossed with White Leghorn, showed that the Langshan and Brahma each carried one pair of dominant genes, while the Cochin and Sultan carried two pairs of dominant genes. A Brahma x Langshan F_1 backcrossed to the White Leghorn revealed that these two breeds may share the same single locus, while a Sultan x Langshan F_1 backcrossed to the White Leghorn suggested that one of the two loci causing leg feathering in the Sultan was shared by the Langshan. Most investigators have suggested that inhibitors to leg feathering are present in some stocks, and they used this idea to explain the appearance of clean shanks where feathering was expected. In Somes' data, only in the weakest shank feathering cross (Langshan x White Leghorn F_1) did 6.7 percent clean shanks appear, where feathered shanks were expected.

It would appear from the data presented here that basically three loci are involved in the types of leg feathering that are considered as breed characteristics. There are two dominant genes at separate loci which when present together produce the heavy leg feathering of the Cochin, Sultan, Belgian d'Uccle Bantam and Booted Bantam. One or the other of these loci by itself produces the weak leg feathering characteristic of the Langshan, Faverolle, and Breda. The slightly heavier expression of leg feathering as seen in the Brahma and Silky may be due to a different allele at one locus as suggested by Somes' work, or it may be due to segregation at the two loci. The Pavloff feathering is due to a recessive gene as suggested by Serebrovsky (1926). It is proposed that the gene symbols *Pti-1*, *Pti-2*, and *pti-3* for ptilopody be assigned to these loci.

Stubs (*Ht, sb-1, sb-2*). Stubs are short partly grown feathers on the shanks and feet that appear in breeds that are normally clean-legged. It is a difficult trait to study since down on the leg at hatch may or may not lead to stubs in adulthood; likewise, clean-legged chicks may develop stubs at a later age.

Serebrovsky (1926) reported that 'heel-tuft' feathering, which is referred to now as 'stubs on hocks' by fanciers, results from a single autosomal dominant gene. It is expressed as small stubs on the inner surface of the shank just below the tibiotarsal joint. It is proposed here that the gene symbol *Ht* for heel tuft be assigned to this locus.

Hays (1943) working with Rhode Island Reds examined 820 offspring at six months of age over eight generations for the more common type of stubs. He concluded that two independent autosomal recessive genes were involved with homozygosity at either locus causing the production of stubs. It is now proposed that the gene symbols *sb-1* and *sb-2* be applied to these two loci. Cumulative effects of these genes, if any, were not determined. A deficiency of females showing the stubs led to the suggestion that the trait may be partially sex-limited.

Two studies indicate a polygenic basis for this trait. By selection, Warren (1930b) established two populations of White Leghorns over three years which differed in the proportions with down on the toes. The high line showed 90 percent while the low line had only 2 percent incidence. F_1 and backcrosses generations indicated that more than one pair of genes was involved. In another study Cole and Hutt (1947) used three full-brothers, none with detectable stubs. One male produced stubs in 40 percent of his daughters, the second produced 13.5 percent and the third produced none at all.

Variations In Feather Length

Crest (*Cr*). A crest is a prominent feature of a number of breeds. Its phenotypic expression varies widely and this may be due to the size and type of comb associated with it. The fullest expression of crest is seen in Crèvecoeur, Houdan, Polish, and Sultan breeds, all of which also have the small V-shaped duplex comb. Crests in these breeds are very large and full, rising well in front of the head and flowing over the back and sides of the head. The crest feathers are similar in shape and texture to hackle feathers. In the Silky breed, which has a walnut comb, the female's crest is similar to those previously mentioned, but the male's crest is medium in size and is as upright as the comb allows, with most feathers streaming backwards and not down the sides of the head. Similarly, the Watermaal breed has a rose comb which extends still further back on the head and its crest is called a tassel. A tassel is described as a tuft or crest of feathers rising from the head at the rear of the comb and falling gracefully over the back of the neck. Other tasselled breeds are the Altsteirer, Breda, Old English Game, Pyncheon, and Sulmtaler Bantam. All of these are single-combed, except the Breda which has no comb, and their tassels are smaller than those of the Watermaal breed. Two breeds, the Appenzeller Spitzhauben and Brabanter, have a very distinctive helmet-shaped crest in which the feathers rise from the rear of the comb and point upward and forward; both of these breeds have the V-shaped duplex comb. The large-crested breeds also have a cerebral hernia associated with their crest of feathers.

Crest was one of the first inherited traits studied in chickens. Hurst (1905) showed that its inheritance was that of an incompletely dominant autosomal gene, and Davenport (1906) showed that only one pair of genes was involved. Dunn and Jull (1927) assigned the gene symbol *Cr* to it. It is not known whether the full crest and the various gradations to the small tassel crest are caused by the same gene. The fact that the Breda, with no comb, has a tassel crest may indicate that a different factor or allele may be involved. The helmet crest associated with the same V-type duplex comb as in full-crested breeds, also seems to be too great a variant to be accounted for by modifying genes; the gene for this type of crest may be at a different locus or it may be a different allele at the *Cr* locus.

Muffs and beard (*Mb*). Muffs, also known as whiskers, consist of tufts of feathers projecting horizontally from both sides of the face, and the beard consists of feathers projecting vertically from under the beak in a downward direction; they always appear together. The muffs and beard trait is a breed characteristic of Houdan, Faverolle, Orloff, Belgian Bearded d'Anvers Bantam, Belgium Bearded d'Uccle Bantam, Crèvecoeur, Dutch Bearded Bantam, Sultan, some varieties of Polish and Silky, and some other breeds. Expression of the trait can be variable in heterozygotes. It is usually identifiable in day-old chicks, but classification may not be totally accurate, particularly with heterozygotes. It has also been shown that presence of the muffs and beard trait is associated with reduction in size of the wattles (Mérat, 1962).

The genetic basis for this trait was studied by Davenport (1906), Serebrovsky and Petrov (1930), and others, and was shown to be caused by a single incompletely dominant autosomal gene. Hertwig (1933) first proposed the gene symbol *Ba*, but Hutt (1949) later proposed that the symbol *Mb* would be more appropriate and the latter is in common use today.

Vulture hocks (*v*). Feathers on the posterior part of the crural feather tract in the area of the tibia are usually soft and fluffy. In the vulture hocks condition, these feathers are changed in structure and length such that they are long and stiff like flight feathers. They project backward in a cluster beyond the tibiotarsal joint and are inclined toward the ground, following the outline of the wings. Vulture hocks is a breed characteristic of Belgian Bearded d'Uccle Bantam, Booted Bantam, Breda, and Sultan. These are all feather-shanked breeds, and although vulture hocks and feathered shanks are believed to be independent traits, Dunn and Jull (1927) and later Jull and Quinn (1931) noted a relationship between the two traits in their crosses. The basis for this relationship is not known, but Danforth (1929) suggested that the vulture hocks gene may merely require the genetic background of shank feathering before it can manifest itself.

Early genetic studies by Davenport (1906), which were later confirmed by Jull and Quinn (1931), demonstrated that vulture hocks results from a single autosomal recessive gene. Hutt (1949) later assigned the gene symbol *v* to this trait. However, Danforth (1929) reported that vulture hocks were feebly indicated in heterozygotes and he felt that the trait was inherited as an incomplete dominant.

Long tail (*Gt, mt*). There are 11 breeds of long-tailed fowl in Japan, the most famous of which is the Tosa-Onagadori, more commonly called Onagadori, Yokohama or Phoenix. Excellent descriptions of these 11 breeds are presented by Tengco and Nishimura (1982). The long-tailed breeds are all believed to have originated from the Shokoku breed, one of Japan's oldest, because of their similarity to the Shokoku in its unusual tail feather arrangement. Oana (1954) explained that the tail of Shokoku type differs from the normal by having additional feathers. These extra feathers consist of a pair of broad long feathers found on the topmost part of the main tail feathers and just a bit below and aslant of the sickle feathers. They grow in the same line with the main tail feathers. Sometimes there are two pairs of extra feathers instead of one.

Females of long-tailed breeds grow feathers at a normal rate and molt them once a year. However, males are different. The male develops a long hackle, and the saddle feathers grow 30 cm per year, while the tail sickle feathers grow at a rate of about one m per year. Males molt their body feathers from time to time but keep about 20 feathers in the tail region, which continue to grow from year to year. Average tail lengths are 3-4.5 m with 11.5 m being a record (Tengco and Nishimura, 1982).

Chamberlain (1900), Cunningham (1903), Davenport (1906) and Bonhote (1914) made some early observations and genetic studies of this trait but their work offered little toward an understanding of it. Sasaki and Yamaguchi (1970) proposed a two-gene hypothesis to explain the difference in the long tail, sub-long tail and short tail breeds. They proposed a dominant gene (*Gt*) which allows for continual growth of part of the tail and saddle feathers, and a recessive gene (*mt*), which makes certain tail feathers and saddle feathers nonmolting. Thus the longtail Onagadori breed would be *Gt*/*Gt mt*/*mt*, a sub-longtail breed, such as the Shokoku whose tail feathers grow about one m per year but are annually molted, would be $Gt/Gt\ Mt^+/Mt^+$, and the ordinary chickens would be $gt^+/gt^+\ Mt^+/Mt^+$.

Environmental factors have an influence on response to these genes. Onagadori are usually raised in small wooden cages, but when raised on the ground and used for breeding the character of their tail feathers changes, growth stops, and the feathers molt readily. When Shokoku are raised in cages, their feathers continue to grow until they are molted in the second or third year (Sasaki and Yamaguchi, 1970).

Eight tail feathers (*ext*). Williams (1979) described a condition he observed in Old English Game and Araucana bantams in which the birds had seven main tail feathers on each side of the tail, plus the main sickle feathers and some extra lesser sickle feathers. Sometimes the extra lesser sickles were not present, and quite often the eight-tail-feather trait was observed only on one side. A normal tail has only six main tail feathers on each side plus the main sickles. This condition appears to be similar to the tail of Shokoku chickens from Japan.

Over a three-year period, Williams (1979) made a number of crosses with this trait, and although his progeny numbers were not large, observations were consistent with the hypothesis that this trait is caused by a single autosomal recessive gene with incomplete penetrance and varied expressivity. He had also observed birds with six and nine tail feathers but did not know if they were associated with the same gene. The gene symbol *ext* is now proposed for the trait.

Surplus flight primaries (*Sf1*, *Sf2*). Normally there are ten primary flight feathers on each wing. Some breeds and strains are characterized by having more than ten. Onishi et al. (1955) found that this condition was fairly common in the Rhode Island Red, Barred Plymouth Rock, and Nagoya breeds, but their White Leghorn and Shamo Games rarely were affected. In studying the inheritance of surplus primaries, they concluded from crosses between Nagoya and White Leghorn that it was due to the complementary action of two completely dominant autosomal genes. They designated the alleles at these two loci as *Sf1* and *Sf2*.

Long Filoplumes (*Lf*). Just before he died, Dr. W.H. McGibbon was working with a feather trait that he called long filoplumes (Somes, 1978a). The filoplumes in the fluff areas of the body were 1-10 cm longer than the adjacent definitive feathers. Somes (unpublished) has also observed this trait in a line of birds at University of Connecticut in which filoplumes project beyond the plumules and contour feathers. Although inheritance studies on this trait are unpublished, McGibbon claimed that this trait was inherited as an autosomal dominant, and he applied the gene symbol *Lf* to it.

Variations In Feather Structure

Feather texture (*Ha*). The texture of feathers differs between breeds and classes of fowl.

Feathering of breeds is usually classified as 'hard' or 'soft' and 'close' or 'open'. Hard-feathered breeds such as the Old English Game, Modern Game, Aseel, Cornish, and Malay have feathers that emerge from the skin at a low angle and thus lay tightly to the body. Feathers of this type are usually shorter and more brittle and snap like a spring when raised. Soft-feathered breeds have soft fluffy feathers that emerge from the skin at a much greater angle and thus give an appearance of openness. The Cochin is an extreme example of this type.

When crosses have been made between hard and soft-feathered breeds, many investigators have found that hard feathering is dominant (Jull, 1940). Wassina (1926) obtained F_2 progeny from Cornish x Pavloff and showed that not only was hard-feathering dominant, but that it segregated as a simple single gene trait. The recessive allele would appear to be the wild-type and must be influenced by modifiers to account for the variations seen among soft-feathered breeds. The gene symbol *Ha* is proposed for this gene.

Frizzling (*F*, *mf*). The frizzled plumage trait has been known for many centuries. It had been reported from many parts of Asia, and Aldrovandi described it in 1600 (Lind, 1963). Today it is recognized as the main characteristic of the Frizzle breed and it is a recognized variety in many bantam breeds.

Although Davenport (1906) was the first to suggest that frizzling was dominant, it was the work of Hutt (1930) and of Landauer and Dunn (1930) that proved the causative factor to be a single autosomal incompletely dominant gene for which the symbol *F* was assigned (Hutt, 1930). Their work dispelled a then currently held idea that the homozygous condition was an embryonic lethal. They later described an autosomal recessive modifier gene which greatly restricted the effects of *F* (Landauer, 1933; Hutt 1936a). This modifier gene received the gene symbol *mf* (Hutt, 1936a). Hutt (1936a) found that the *mf* gene was quite common and was in most nonfrizzled fowl that he tested.

Because of the interaction of these two genes, there are four phenotypic expressions of frizzling, only one of which is of interest to the show fancier. Unmodified homozygotes exhibit extreme recurving of the rachis of all feathers with extreme curling of the barbs. Feathers are very narrow and break off easily so that these birds are usually quite bare. The modifying gene lessens the extreme aspects of the homozygotes so that they appear less woolly. The unmodified heterozygote is the fanciers show bird; feather shafts and barbs are recurved, but to a much lesser extent than with the homozygote, and the rectrices and remiges are much less affected. The *mf* gene modifies heterozygotes so that some birds are almost indistinguishable from the wild-type.

The action of the *F* gene has been shown to be localized in the feather follicle and does not result from a metabolic disorder (Landauer and Aberle, 1935). It is also not due to the presence of different proteins, but to changes in the spatial distribution of common structural elements (Brush, 1972). A number of studies have been undertaken to determine the physiological effects of this gene on the bird. Basal metabolism of frizzled birds is accelerated, leading to increased production of both thyroid and adrenal gland hormones. Food intake, oxygen consumption, heart rate, and volume of circulating blood are increased. As a result of this, it is common to find enlargement of the heart, spleen, gizzard, and alimentary canal in frizzled birds (Benedict et al., 1932: Boas and Landauer, 1933, 1934; Landauer and Aberle, 1935; Landauer and Upham, 1936).

Silkiness (*h*). The gene for silkiness has also been known for many centuries. It was mentioned by Marco Polo in 1298-99. It was later mentioned by Aldrovandi in 1600 and is illustrated in his book as the wool-bearing hen (Lind, 1963). This condition is a major characteristic of the Silky breed.

The silky trait radically changes the structure of all feathers. In general, feather shafts are more delicate with unusually long and frequently bifurcated barbs. The barbules are elongated, irregularly arranged, and lacking in both distal and proximal barbicels. Thus the feathers do not have a flat web and the barbules are not held together. All of this results in a silky or woolly appearance. The rectrices are more affected than the remiges.

Inheritance of silkiness has been studied by many (Davenport, 1906; Cunningham, 1912; Bonhote, 1914; Jones, 1921; Dunn and Jull, 1927) and all showed that it is caused by an autosomal recessive gene. Jones (1921) collected data on silkiness in nine different breeds and showed them all to be genetically the same. The gene symbol *h* for hookless was first given to this trait by Dunn and Jull (1927).

Because thyroidectomy results in the production of silky-like feathers, the relationship between the thyroid and silkiness has been studied. Since regenerating silky feathers were not innately refractory to excess thyroxine injected into silky birds (Juhn, 1956), and thyroxine secretion rates did not differ between silky and nonsilky birds, the action of the *h* gene must be at the feather follicle level (Juhn and Bates, 1960). Protein electrophoretic patterns of silky feathers indicated that the differences observed were not due to the presence of different proteins, but to changes in the spatial distribution of common structural elements (Brush, 1972).

Sex-linked naked (*n*). Hutt and Sturkie (1938) reported on the first of several naked mutants, this one originating from a White Leghorn flock. Naked chicks varied considerably in degree of nakedness at hatching time. In general the anterior end of the bird was more naked than the posterior. In extreme cases the few down feathers present were wiry, short, and thickened because the barbs failed to separate. As juveniles, the birds became quite naked as the down feathers wore off; replacement feather growth was extremely slow. Some feather follicles did not erupt, whereas others were swollen and contained dried blood. In some cases, feathers would break off near the skin. Adults were more completely covered than juveniles, but even among these the variation in coverage was wide. Pterylosis was normal and all pterylae contained some feathers, and there were down feathers in the apteria. The remiges were usually all missing, but some stubs remained. Up to four rectrices were present. In general adult birds had a rough appearance. Limited data indicated that naked birds did not radically differ from normal birds in growth, age to sexual maturity, or in egg characteristics.

Hutt and Sturkie (1938) showed that naked resulted from a single sex-linked recessive gene to which they assigned the gene symbol *n*. They also showed that the *n* gene was semilethal both before and after hatching; 46.7 percent of naked embryos died at 19-21 days of incubation and 55.3 percent of those hatching died by six weeks of age. Hemizygous females and homozygous males were equally affected. Attempts to localize the feather defect by means of skin transplants and the feeding of cysteine and cystine led to the conclusion that the mutation primarily affected the feather follicles.

Wing patch. Jeffrey (1985), while breeding Self Blue Old English Game bantams, produced a cockerel which had what he called a wing patch on the bows of both wings. The wing patch was an area of 10-15 mm^2 located on each wing bow, in which the feathers never developed beyond the pin feather stage. The area adjacent to the patch contained feathers of intermediate size which looked like silky feathers. When the bird molted, the pin feathers also molted, but were replaced again with pin feathers. Banning-Vogelpool (1983) cited a report in the Belgian periodical *Aviculture* of September 1971, describing a defect in Porcelain Booted Bantams in which there is a malformation of the feathers of the wing bow, which do not molt normally but stay silky-like. In both of these cases, the wing patch trait was observed in birds homozygous for the self blue gene (*lav*).

When Jeffrey (1985) crossed his self blue male with the wing patch to a self blue sister, he produced five male progeny all with wing patches and six female progeny all with perfectly normal wing bows. No additional matings were made. Although the genetic basis of this trait is still unknown, it would appear that it is inherited, its expression is limited to males, and it may possibly be linked to the lavender locus (*lav*).

Abnormal feathering (*af*). An interesting abnormal feathering trait has been described from Great Britain by Bulfield (Somes, 1988). Feathering in chicks was normal, but juvenile and adult feather structure was not, and its distribution was patchy. This trait was very variable in its expression with extreme cases being completely naked; however, some of these birds did develop secondary flight feathers as adults. The inheritance of this abnormal feathering trait was that of an autosomal recessive and the gene symbol *af* has been proposed for it.

Alopecia. The DAM chicken line developed at University of Massachusetts was established from a single female of their 'Brown Line' genetic stock. The DAM line is characterized by high incidence of postnatal amelanosis and blindness, and a low incidence of autoimmune thyroiditis and a feathering defect (Smyth et al., 1981). The first three of these characteristics are described in other chapters.

The feathering defect, which came to be known as alopecia, first appeared in a G_2 generation female with two more females showing the trait in the G_3 generation. By the G_5 generation, the incidence was up to 2.8 percent. Chicks appeared normal at hatching, but by 6-8 weeks of age they developed a structural defect in the rachis and barbules at the tips of the remiges and rectrices. Feathers developing later showed varying degrees of delayed development. Many feathers broke off on older birds, leaving patches of baldness and total denudation in severely affected birds. Birds with alopecia always showed some degree of feather amelanosis, and although both sexes were affected, males were less likely to show extreme denudation. Histological and histopathological examination of defective feathers showed abnormal barb ridge development, pulp cell hyperplasia, and a pronounced infiltration by a mixture of heterophils, mononuclear cells, and plasma cells. It was hypothesized that normal feather development was interfered with by hyperimmune activity of the DAM line (Jerszyk et al., 1983).

After two generations of selection, the incidence of alopecia was increased in a subline from 0.6 to 17.4 percent. Specific genes responsible for this trait were not identified, but outcrossing to normally feathered birds produced all normal birds, indicating that its inheritance was recessive (Jerszyk et al., 1983).

Dysplastic remiges (*dr*). This plumage abnormality (Figure 6.2) was first observed at University of Saskatchewan in a synthetic line (Urrutia et al., 1983). Affected adults lacked a variable number of remiges and rectrices and in extreme cases wing coverts were also affected. Expression of the trait was first evident in the centre of the wing and tail. The mildest expression resulted in the loss of only the axial feather. More extreme expressions resulted in an increasing lateral loss of primary and secondary feathers with the most severe expression being total flight feather loss. Tail feather losses corresponded to the severity of the syndrome in the wings, but the number of affected rectricial follicles was always lower.

Affected follicles were either empty, contained a mass of feather material, or they contained a deformed feather. This condition was not evident in day-old chicks, but started to show itself in the remiges by the 6-16 days of age and in the rectrices by 21-28 days of age. Each feather follicle affected in the juvenile plumage was also affected in the adult plumage. It was subsequently shown that the defect is also expressed in the crural tract of birds with the vulture hocks (*v*) trait (Ellis and Crawford, 1988, personal communication).

Two generations of breeding tests showed that the abnormality was inherited as a single autosomal recessive, with the gene symbol *dr* being assigned to it. Segregation data were based on classifications at four weeks of age, at which time expression in *dr*/*dr* birds was about 96 percent certain. Even so, there was no doubt about the inherited basis of this plumage abnormality.

Flightless (*Fl*). The flightless trait was studied by Warren (1933b). In this condition flight feathers, tail feathers, and other large feathers had a shaft defect causing them to break when subjected to pressure. Feather breaking began as soon as the feathers were mature. Small body feathers generally did not break, but as plumage became older there was a tendency for birds to become progressively rougher in appearance. The flightless condition could be recognized in young chicks by one month of age, with flight feathers being irregular in length and some even being broken by this age. Shafts of the flight feathers were very rough and scaly. Chemical studies (Marlow and Caldwell, 1934) showed that cystine content of flightless feathers was 18.5 percent less and phosphorus 124 percent greater than in normal feathers; total sulphur and nitrogen contents were similar. X-ray analysis showed defective feathers to be only slightly fibred compared to normal feathers. The micelles of normal feathers are long and narrow and lie parallel to each other and the feather axis, while in defective feathers the micelles showed only a trace of orderliness. The physiological basis for this trait would appear to be an abnormality of protein metabolism.

Based on F_1 and backcross data, Warren (1933b) reported that flightless was due to a single dominant gene. Warren (1937) modified his conclusions after studying the F2 generation in which there were normal, flightless, and a few featherless birds. The ratio of normal to flightless was about 2:1, and it was assumed that the featherless birds were homozygotes, but this was not tested because featherless birds never reached sexual maturity. Thirty-one flightless F_2 birds were test-mated and all proved to be heterozygous. Featherless birds were all normal appearing at hatching, but by four weeks of age they could be classified as featherless. At this age they also began to show growth retardation, and deformities of the beak and toenails, which broke easily. It appeared that the homozygous state was lethal and that 67 percent of the homozygotes did not survive to four weeks of age. Exact time of lethality was not determined, but hatchability was only slightly reduced. It may be that some died during embryonic development and others during the first few weeks of life. From these F_2 data, it was evident that the flightless gene was incompletely dominant and generally lethal in the homozygous state. The gene was assigned the symbol *Fl* by Hutt and Lamoreux (1940).

Fray (*fr*). Warren (1938) reported a feather variation characterized by a frayed appearance of the wing flight and tail feathers. In real extreme cases, body feathers were also lacking in smoothness, but generally only wing and tail feathers were affected. The frayed appearance was due to a defect of the barbules and hooklets, particularly the anterior ones, which resulted in imperfect interlocking of the anterior and posterior barbules. The separated barbs gave a frayed appearance to the vane, and the trait was so named for its appearance. The frayed condition was not recognized at hatching time and was generally difficult to recognize in juvenile plumage.

Originally two frayed females were used to establish a flock carrying this trait. Subsequent crosses showed that the frayed trait was inherited as a simple autosomal recessive. A deficiency of frayed individuals in backcrosses may have been due to lowered viability in frayed chicks or the inability to recognize frayed individuals with extremely mild expression of the trait. The gene symbol *fr* was assigned to this trait.

Hypoplasia of tail feathers (*Hy*). A trait which superficially resembles rumplessness is characteristic of the Ingie breed, found only on Tanegashima Island in Japan (Hashiguchi et al., 1978). It has been maintained pure since its initial introduction from England in 1894. All Ingie fowl are characterized by peculiar rectrices and other associated tail-region feathers and appear to be rumpless. However, X-ray examination showed that they possess the normal uropygial gland, free caudal vertebrae, and pygostyle, and thus are not rumpless. Although the numbers of tail coverts, main sickles, lesser sickles, and main tail feathers are similar to normal birds, their structure is quite different. All these feathers appear extremely under-developed, and they are all frizzled, very thin and long, with a slender rachis. The anterior and posterior barbules are also abnormal and there is a deficiency of hooklets.

Crossbreeding tests between Ingie fowl and White Leghorn and White Plymouth Rocks showed that this hypoplastic trait is inherited as a simple autosomal dominant, and the gene symbol *Hy* was proposed.

Matted down. This condition appeared in northern Germany in a White Leghorn flock (Gleichauf, 1965). The chicks had a bristly singed appearance, resembling the ropy, stringy, and sunsuit mutants. Chicks looked stunted, but they were in fact only slightly lower in body weight (1.5 g) than the breed average. The condition was designated matted down. The matted appearance was due to sticking together of the ends of the down plumules and failure of the sheath to split during the drying process. The elements of the down feather were not changed in either structure or arrangement, but were just not able to be normally released from the sheath. Histomorphological studies during embryonic development showed that this down abnormality was caused by hyperkeratosis of the feather sheath at 16-17 days of incubation.

Genetic analysis of matted down indicated that it had a polygenic basis, with a number of additive genes being involved. Normal down formation relative to matted down was shown to be only incompletely dominant. There also appeared to be a relationship between matted down and the formation of pigmented head spots in White Leghorn chicks. Continued selection for large head spots resulted in chicks whose down structure closely resembled that of the matted down strain.

Ragged wing (*rw*). This feather defect was discovered independently at New York and Kansas Agricultural Experiment Stations and was described in a joint publication (Hutt et al., 1944) as a mutant feather condition called ragged wing (Figure 6.2). Ragged wing must be fairly widespread for additional reports of presumably the same trait have appeared in the literature as ragged wing (Potemkowska et al., 1977), as stumpy wing (Gleichauf, 1961), and short wing (Morgan, 1963).

The ragged wing condition was not evident in day-old chicks or even in juveniles, but became evident in the adult plumage. Juhn and Shaffner (1962) observed that selection for the condition changed its time of expression to an earlier age and caused a greater number of follicles to be affected. They were also able to show sire family differences. In its most extreme expression, the ragged wing condition caused a flightless bird because of the loss of all flight feathers. However, total loss of remiges was not the usual case. Generally some flights were missing but others were only shorter in length. The opposite extreme was that all remiges were present but they were shorter than normal. Tail feathers were never affected regardless of the degree of remex abnormality. When regeneration was accelerated by feather removal, feather papillae gradually lost their ability to regenerate a feather; by day 21 most regeneration was sharply diminished. A very early embryonic determination was suggested, resulting from a developmental dissociation of the ectodermal and mesodermal components of the feather germ. Severity of the dissociation increased with posthatch age of the chick (Juhn and Shaffner, 1962).

The mode of inheritance was studied at both experiment stations, and very similar results were obtained (Hutt et al., 1944). Data clearly showed that the trait was inherited as an autosomal recessive, but presumed modifying genes prevented full expression. At both stations about half of the homozygous birds did not express the trait. To further show the effect of modifiers, the same ragged wing males were mated to ragged wing, short wing, and phenotypically normal ragged wing females; 39, 24 and ten percent of their offspring respectively were ragged-winged. The ragged wing trait was assigned the gene symbol *rw* at a later date (Somes, 1980).

Porcupine (*pc*). On page 352 of *Aldrovandi On Chickens* (Lind, 1963) is pictured what is presumably the first reported porcupine fowl. Waters (1967) reported recurrence of this mutant. Feathers of porcupine chickens did not develop in a normal fashion, but instead appeared as spiny quills, even in day-old chicks. The condition was identifiable at the beginning of embryonic feather development. In adults the feathers still remained underdeveloped and quill-like. Porcupine chickens were difficult to raise, needing additional heat to counter excessive body heat losses. Adult hens layed very few eggs and fertile eggs were never produced, even when artificial insemination was employed.

The only breeding data available consisted of two years of matings between heterozygous males and females. Data from both of these years from these F_2-type matings were very consistent with the hypothesis that this feather condition was caused by a single autosomal recessive gene to which the gene symbol *pc* was assigned.

Ropy (*ropy*). 'Sticky' newly-hatched chicks which failed to dry to their usual fluffiness were observed by Warren (1949). Identification at hatch was made easy by the marked contrast with normal chicks. As adults, the birds superficially appeared to be normal, but they had a fissure in the proximal half of the large wing feathers which was only visible by examination of the underside of these feathers. Sometimes body feathers showed a slight roughness, which led to the trait being called ropy. Ropy birds appeared to be normal in all other aspects.

Genetic crosses showed that this feather defect was the result of a single autosomal recessive gene which was later given the gene symbol *ropy* (Somes, 1980). Because of its similarity to a stringy mutant (stringy-2) being studied at the same time (Buss et al., 1950), ropy and stringy birds were crossed to determine if they were part of an allelic series. All 59 offspring from the test cross appeared normal, both as chicks and as adults. The genes that were controlling these two traits were presumed to be located at different loci.

Stringy (*st*). A variant in down feathers, which became known as stringy, was reported by Kessel (1945a). The down plumage was stringy in appearance because the down barbs

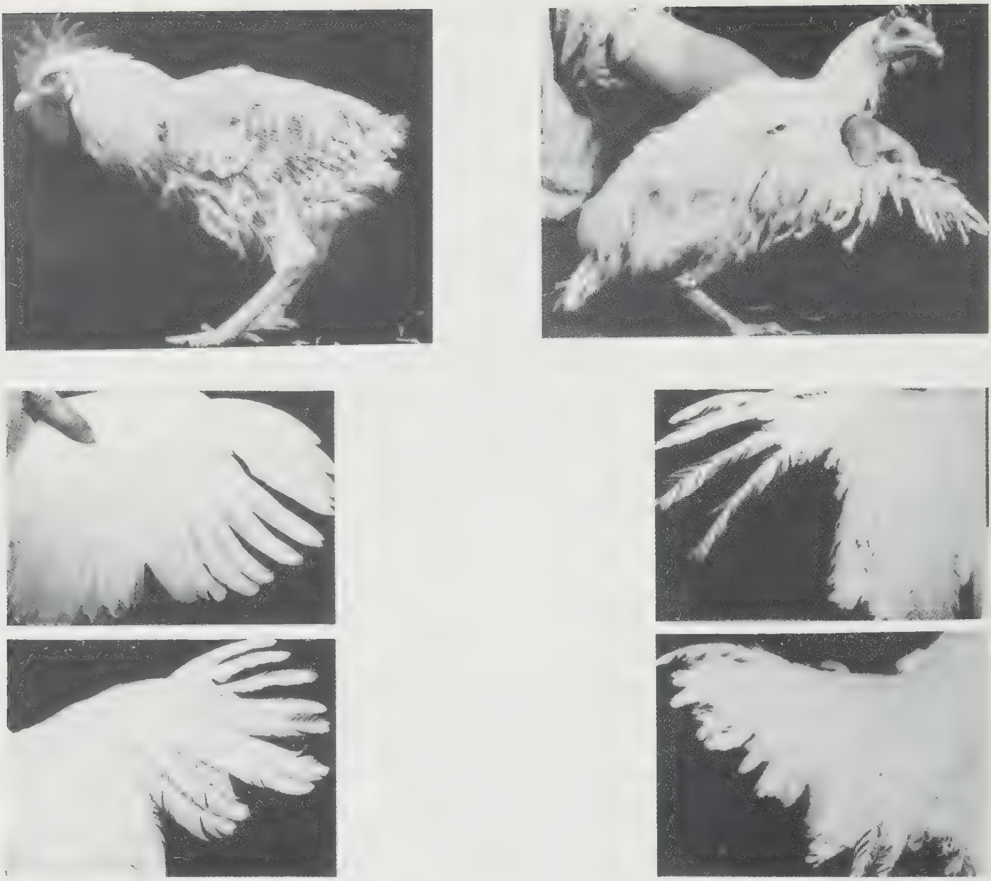

Figure 6.2. Feather structure variants. Upper left — sunsuit (*sn*) (from Hutt and Long, J. Hered. 41:146, 1950). Upper right — stringy-2 (*st-2*) (from Buss et al., J. Hered. 41:142, 1950). Lower left — normal and ragged wing (*rw*) (from Hutt et al., J. Hered. 35:30, 1944). Lower right — intermediate and severe forms of dysplastic remiges (*dr*) (from Urrutia et al., J. Hered. 74:102, copyright 1983 Am. Genet. Assoc.).

were twisted and stuck together. The condition was most abundant on the back in front of and between the wings. At hatching the affected chicks were full size and very active, but by one week of age they began to weaken. All had died before two weeks of age.

The parents of these abnormal chicks were a male and his seven sisters. They all had normal plumage, but 14 of the 85 chicks raised had stringy down feathers. The mode of inheritance would appear to have been recessive, but it is uncertain whether this trait was autosomal or sex-linked. The gene symbol *st* was later assigned to it (Somes, 1980).

Stringy-2 (*st-2*). An interesting feather abnormality was studied simultaneously at the Purdue and Kansas Agricultural Experiment stations. A check on sources of the two stocks indicated that they had a common origin. Subsequent crosses between the stocks proved that they carried the same mutation and it was reported jointly (Buss et al., 1950).

The stringy trait was named after the characteristic appearance of adult birds in which most of the barbules were absent from wing and tail feathers. At one day of age chicks were 'sticky'; the down feathers were not fluffy, and they were parted down the middle of the back. Adult wing and tail feathers were generally short, and the barbs did not spread out to form the vane. The sheath also persisted for longer than normal. In addition to feather abnormalities, there was also beak and toenail involvement; the premaxilla and the toenails were longer than normal and quite often the beaks were crossed (Figure 6.2). It was suggested that this condition might be manifesting itself by modifying the amount or toughness of keratin produced in the epidermis. Adult birds of both sexes had reduced vigor. Although males were very poor semen producers, female egg production was equal to that of normal birds.

Crosses at both stations, as well as a cross between the two sources, demonstrated that only one trait with a common basis was involved. Its inheritance was determined to be that of a single autosomal recessive to which the symbol *st-2* was later assigned (Somes, 1980).

Sunsuit (*sn*). A most unusual feather abnormality was reported by Hutt and Long (1950). Expression varied among individuals, but in general the extremities were bare of feathers while the body was better covered. The name sunsuit was given to the type specimen of this condition (Figure 6.2). In day-old chicks, the down plumage appeared wiry and sticky, particularly over the back and neck. The wiry-sticky appearance was caused by barbs of the down plumules being stuck together at their tips. As juvenile plumage developed it was also abnormal, but not nearly so much as in the adult. Juveniles did not lack any of their primary flights, but the primaries were ruffled and frayed and appeared very much like frizzled feathers. In the adult, the condition was quite extreme. The remiges were reduced in number and length. The tail feathers were also reduced in number and length, but not to the extent of the flights. Feather webs failed to break from their sheaths, so that affected areas were covered with quills rather than with feathers. Not all feather tracts were equally affected however. The head and neck were usually normal; the posterior spinal, lateral, and crural tracts were intermediate; the humeral, alar, and femoral tracts showed the greatest degree of abnormality. Males were generally more affected than females. New feathers seemed to be generated continually. Most birds at maturity also showed abnormalities of the beak and toenails. Upper and lower beak were elongated, with the upper beak being sometimes arched or crossed. The toenails became excessively long with age. These abnormalities suggested that the underlying defect associated with this mutant was some kind of dyskeratosis.

Genetic studies of the sunsuit trait demonstrated that it was inherited as a single autosomal recessive. The gene symbol *sn* was assigned to it. There was no lethality associated with this gene.

Wiry (*wi*). The wiry syndrome in day-old chicks was described by Fiser et al. (1973). The general appearance was due to sticking together of the down feathers which also lacked barbs on their distal halves. As adults, the most affected feathers were the wing flights and the tail feathers. The barbs of wiry feathers adhered together and to the feather shaft so that each feather was partially covered with the sheath and the barbules were only 15 percent as long as normal ones. However, as affected birds became older their appearance became less distinguishable from that of the normal birds. Histologically the barb vane ridge of the wiry feather appeared to be more closely spaced and to develop in a corkscrew fashion when compared to the normal. The mesodermal pulp was also less cellular than usual.

Fiser et al. (1975) examined the effect that brooding temperature had on wiry chicks. Hatchability records showed that the wiry gene had no adverse effects during incubation, but that wiry chicks had to be brooded at 37° C for the first two weeks in order to keep mortality at a minimum. When brooded at 32-33° C, 60 percent of wiry chicks died by the second day and 88 percent by the sixth day; only three percent of normal chicks died during the same period. When exposed to field conditions (25° C) and not permitted to huddle, all of the wiry chicks died within 24 hours, whereas none of the heterozygous normal chicks died. Thus the wiry trait was a potential facultative lethal when brooded under normal conditions and an obligate lethal when exposed to field conditions.

Genetic studies showed that the wiry condition was the result of a single autosomal recessive gene with ratios being very close to those expected (Fiser et al., 1973). The gene symbol *wi* was assigned to this trait by Somes (1980).

Woolly (*wo*). Jones and Kohlmeyer (1950) first reported this unusual feather mutation and Jones and Morgan (1956) amplified on its characteristics and inheritance. At one day of age chicks showed considerable variation in expression of the woolly condition. There was a pronounced shortening of the down plumage in all chicks and completeness of feathering varied from normal to seminaked. When down feathers were lacking, it was usually the dorsal and abdominal tracts that were affected. Varying amounts of down feathers were clubbed or rolled into the shape of small feather nodules. Another distinguishing feature of day-old chicks was the slick shiny appearance of their feet and legs, particularly on the bottoms of the feet and the back of the shanks. As juveniles and adults, feathers of woolly birds were normal in quantity, distribution, and length, but all feathers were abnormal in structure, being rough and incomplete and giving the appearance of lacking smoothness and symmetry. Distal barbules were aligned parallel to the barb and proximal barbules were absent from woolly feathers so that normal interlocking of barbules to form a smooth feather was not possible (Swanson and Morgan, 1956). Amino acid content of woolly feathers was not found to differ from that of normals (McRoberts, 1968). Affected birds had enlarged uropygial glands (Swanson and Morgan, 1956). Woolly birds in general did not perform as well as normals (Jones and Morgan, 1956). Although fertility and hatchability were normal, usually twice as many woolly embryos were dead-in-shell at 19-21 days than normals. They were less active at hatching, needed more heat, and showed twice as much mortality by two weeks of age. Females were slower to reach sexual maturity and both sexes were about 25 percent smaller in body size.

A series of genetic matings showed that the woolly trait was inherited as a single autosomal recessive with complete penetrance. Jones and Morgan (1956) assigned the gene symbol *wo* to this feather structure mutant.

Henny feathering (*Hf*). In most chicken breeds adult feather structure is sexually dimorphic. The hackle, saddle, sickle, back, and wing bow feathers of males are long, pointed, and fringed. This type of feather structure is known as cock feathering. Female feather structure by comparison is rounded, with solid vanes, and is known as hen feathering. The presence of the female hormone estrogen results in hen feathering while cock feathering is independent of gonadal hormones (Domm, 1939). Sebright bantams and some strains of Campines are characterized by a trait known as henny feathering. Occasionally this trait is also found among Hamburgs, Silver-laced Wyandottes and Games (Hutt, 1949). Henny feathered males of these breeds are indistinguishable from females in their feather structure.

Early studies on the physiological basis of the henny feathering trait were summarized by Hutt (1949). More recently, the biochemical pathogenesis of this trait has been elucidated. George and Wilson (1980) and Leshin et al. (1981) have shown that the rate of peripheral estrogen formation, measured as aromatase activity, is greatly increased in henny feathered males and that testosterone, but not dihydrotestosterone, is the androgen being converted to estradiol (George et al., 1981). It is hypothesized that henny feathered birds synthesize an increased amount of a structurally normal ovarian-type aromatase in peripheral tissues (Leshin et al., 1983) with the defect being due to some regulatory mechanism (Somes et al., 1984).

Early genetic studies of this trait showed it to be autosomal and dominant. Morgan (1919, 1920) proposed two genes, but Punnett and Bailey (1921) showed later that a single incompletely dominant gene was the causative factor. Punnett (1937) proposed the gene symbol *H* for this condition, but this was later changed to *Hf* (Hutt, 1949). Punnett (1937) also proposed that the *Hf* gene was normally carried on the Y chromosome of ordinary breeds, but that it had been translocated to an autosome in henny feathering breeds. Somes et al. (1984) verified the single dominant gene basis of this trait by following the segregation of skin aromatase activity. Because F_1 progeny have half the enzyme activity of the Sebright, they were able to show that even though the gene was incompletely dominant at the biochemical level, there was sufficient enzyme to allow for full hen feathering to develop in Hf/hf^+ males and thus henny feathering was transmitted as a dominant trait. Skin aromatase activity segregation in the F_2 generation of a Sebright x Campine cross showed that the henny feathering trait of both of these breeds was caused by genes at the same locus (George, 1988, personal communication).

Variations In Growth Rate Of Feathers

Growth of normal feathers. In general, at time of hatch only the primary and secondary feathers are evident, the rest of the chick being covered with down feathers. In a gradual and orderly fashion the down feathers are replaced by chick contour feathers. This usually takes 4-8 weeks. These feathers in turn are replaced by juvenile plumage, which is in turn replaced by adult plumage. Plumage is molted and replaced on a yearly basis. Molting and feather growth are influenced by many factors, both environmental and genetic. Only the genetic factors will be discussed here.

Sex-linked slow feather growth (K^n, K^s, K). Asiatic, English and American breeds of chickens are characterized as slow feathering breeds, while the Mediterranean breeds as well as some others are characterized as being rapid feathering breeds. Serebrovsky (1922) determined that a single pair of sex-linked genes was responsible for this difference with the mutant gene for slow feathering being dominant to the wild-type for rapid feathering. Warren (1925) verified this important discovery, and many others have since studied and reported on this sex-linked locus. The *K* gene symbol designation was proposed by Hertwig and Rittershaus (1929). Somes (1969) reported a delayed feathering mutant in which feathering rate was extremely slow and which he showed to be a third allele at the *K* locus; this gene was dominant to both *K* and k^+ and was designated as K^n (Figure 6.3). McGibbon (1977) reported on yet another allele at the *K* locus. He termed this mutant 'slow' to distinguish it from Warren's (1925) 'early' and 'late' alleles, and gave it the gene designation K^s. The order of dominance of these alleles thus became $K^n>K^s>K>k^+$.

The original rapid and slow phenotypes were shown in studies by Warren (1930a) to differentiate with a high degree of accuracy at hatching time and much has been written about their use for autosexing. The primary and secondary feathers of day-old rapid feathering chicks project well beyond the down and wing coverts, while those of slow feathering chicks do not. With the K^s and K^n mutants, there is practically no primary and secondary growth and the coverts are very short. At 12 weeks of age, birds homozygous or hemizygous for k^+, K and K^s are usually completely feathered, although some K^s/K^s males may have no main tail feathers (McGibbon, 1977). By contrast, birds carrying the K^n gene never do completely feather out. Females and homozygous males are usually naked during the juvenile stage. As adults, females may vary from completely naked to almost completely feathered; adult males usually have the ventral tract bare of feathers and the rectrices and remiges are absent; heterozygous males develop feathers earlier than homozygous males and usually appear quite normal as adults except for the absence of rectrices. The K^n gene also had a pleiotropic effect on body organs other than feathers (Somes, 1975). Comb development was greatly delayed and in females it never reached normal size or color even when sexually mature. By contrast, the uropygial gland was extremely hypertrophied with significant differences in the amounts of individual fatty acids in its lipid content (Walker and Somes, 1978). Environmental temperature appeared to have no effect upon the gland size or lipid composition, but hot environments allowed comb size to approach normal (Walker and Somes, 1979).

A very large body of literature exists concerning the k^+ and K alleles of this locus and their influence on growth and reproductive traits. These relationships are discussed in Chapter 20.

Retarded-tardy feather growth (t^s, t). Among stocks known to be rapid feathering (k^+/k^+ and $k^+/-$), not infrequently there will appear chicks which do not grow feathers at the expected rate. Usually such birds are culled. Warren (1933c) studied late feathering Leghorns of this type. The 'retarded' birds, as he called them, were normal as adults, but as chicks they were very distinct from the normal rapid feathering chicks. At one day of age, retarded chicks had six well-defined primary flight feathers but only the first three secondaries were well-defined, while rapid feathering chicks had six of each. Tail feathers appeared in rapid feathering chicks by ten days of age, but not until three weeks of age in retarded chicks. By six weeks of age, retarded chicks appeared the same as rapid feathering ones.

McClary and Bearse (1941) briefly described an autosomal recessive slow feathering condition in White Leghorns. Later, McGibbon and Halpin (1946), and Jones and Hutt (1946) simultaneously reported on studies of extremely slow feathering White Leghorns which were known to be of the sex-linked rapid feathering genotype. Their 'tardy' chicks were more delayed in feathering than were the retarded chicks of Warren (1933c). At hatching, tardy chicks had the normal six primaries but no secondaries, and at ten days of age there was still little development of the flights, which were narrower and shorter than normal. Tail feathers did not appear until about eight weeks of age and even then they developed slowly. At maturity, retarded and tardy birds could not be distinguished from each other or from normal rapid feathering birds. The best ages at which to classify these two types of chicks was ten days of age for retarded chicks and prior to eight weeks of age for tardy chicks (Figure 6.3).

Genetic studies of Warren (1933c) showed that the retarded condition was caused by a single autosomal recessive and that the birds had to be of the sex-linked rapid feathering genotype (k^+/k^+ and $k^+/-$) for the gene to express itself. Genetic studies by Jones and Hutt (1946) on the tardy trait showed that it too was caused by a single autosomal recessive and that it was a recessive allele to retarded. Thus an autosomal multiple allelic series was identified which modified the expression of sex-linked rapid feathering birds. Jones and Hutt

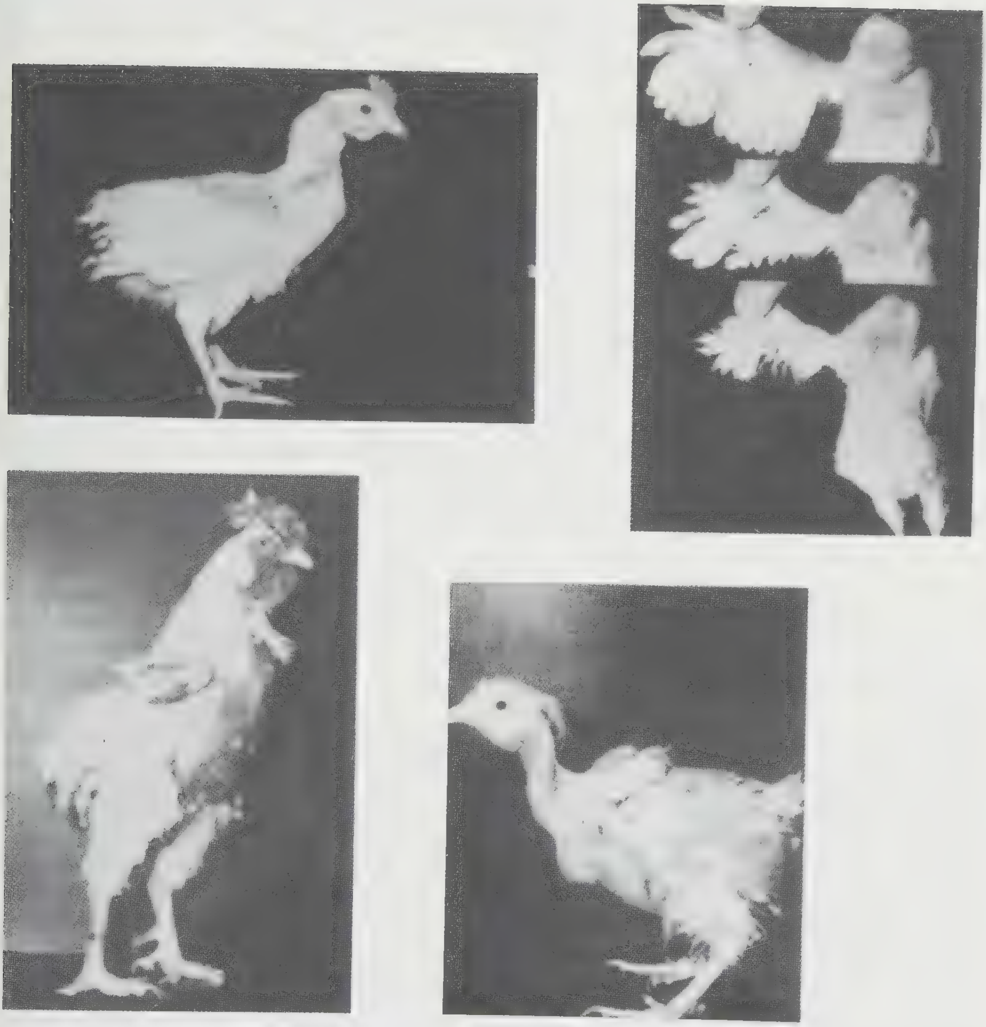

Figure 6.3. Feather growth variants. Upper — eight-week-old tardy (t/t) male, and normal (T/T), retarded (t^s/t^s), and tardy (t/t) chicks at ten days of age (from Jones and Hutt, J. Hered. 37:198, 1946). Lower — delayed feathering male (K^n/K^n) and female (K^n/-) (from Somes, J. Hered. 60:283, copyright 1969 Am. Genet. Assoc.)

(1946) assigned the gene symbols T^+, t^s and t to these alleles. The superscript s for retarded was used to indicate its effect on the secondary flight feathers.

Modified slow feathering. Smyth and Classen (1976) reported a polygenic trait which modified the phenotype of K-type slow feathering birds. The primary flight feathers were reduced in length at hatching time, but the coverts were not similarly shortened. Superficially, these chicks appeared to be rapid feathering and this would lead to sexing errors if this trait was present in a stock which was to be feather-sexed. There was also a dosage effect with K/K males having a higher incidence of the modified slow feathering than K/k^+ males.

Other Plumage Variants

There are a number of characteristics which the fancier considers as defects or deformities and which probably have a genetic basis. Such traits as wry tail, split tail, squirrel tail, drooping tail, split crest, split wing, slipped wing, and twisted feathers would fall into this category. The inherited bases of these conditions have not been reported, but most if not all are probably recessively inherited. Somes (unpublished) made crosses between wry-tailed and normal-tailed birds and all F_1 progeny were normal-tailed.

Tail carriage varies considerably among chicken breeds and thus is an inherited trait with probably more than one locus being involved. Drooping and squirrel tails are defects for most poultry breeds, but they are the breed characteristics for others. The Japanese Bantam has an extremely high tail. Aseel, Cornish, Cubalaya, Malaya, and Shamo breeds all have tails that are described as drooping or carried below the horizontal. None of these extreme tail carriages has been studied genetically.

VARIANTS OF SKIN AND RELATED STRUCTURES

Variations In The Comb

Single comb. Single comb is the wild-type as far as genetic studies are concerned (Figure 6.4). It is the comb type of three junglefowl species (*Gallus gallus, G. sonnerati,* and *G. lafayettei*). It consists of a single blade which runs the length of the head and is topped by a varying number of points.

The size of a single comb varies among breeds, but it is consistent within breeds, and thus probably under genetic control. Many single comb breeds, particularly the heavy ones, have relatively small single combs; the Mediterranean breeds as well as some others have much larger single combs, some so large that they lop over. Axelsson (1933) quantitated this difference when he showed that the mean area of a Rhode Island Red comb was only 389 mm^2, while that for White Leghorns was 1,321 mm^2. It has also been shown that the area of the comb is about three times as variable as its length (Axelsson, 1933; Pearl and Pearl, 1909).

Five points on a single comb is the standard, but variations above and below this number are not uncommon. Axelsson (1933) found a correlation of 0.98 between the number of comb points in sires and their daughters which seems to suggest that multiple factors are involved. The He^+ locus, a modifier of rose comb, has also been shown to affect single comb point numbers.

Spike blade comb (sb). Warren (1939) described a variation of single comb in which the comb ended in a single spike instead of the usual broad oblong blade. Birds could not be classified for this trait with a high degree of accuracy until they had reached sexual maturity. Genetic crosses made by Warren (1939) showed that this trait was inherited. The trait was recessive and not sex-linked, but segregation irregularities seemed to indicate that a small percentage of heterozygotes could exhibit the trait and that it was sometimes suppressed in a small percentage of homozygotes. Females were better able to express the

Table 6.2. Listing of variants of skin and related structures in chickens.

Trait and gene symbol		Trait and gene symbol	
Variations in the comb			
Single comb	+	Pea comb	P, p^+
Spike blade	sb, Sb^+	Walnut comb	$P + R$
Side sprigs	*	Duplex comb	D^v, D^c, d^+
Multiplex	*	Breda comb	bd, Bd^+
Rose comb	R, r^+		
Smooth	he^l, He^+		
Trifid	?		
Variations in the uropygial gland			
Uropygial	U, u^+	Double gland papillae	dgp, Dgp^+
Cleft and double papillae	$U\text{-}2, u\text{-}2^+$		
Variations in the spur			
Spurlessness	sl, Sl^+	Multiple spurs	M, m^+
Auxiliary spur	As, as^+	Molting spur	?
Double spur	ds, Ds^+	Spurs in females	?
Variations in the skin			
Dactylolysis	dac, Dac^+	Avian ichthyosis	$dehy, Dehy^+$
Blistered foot lethal	bf, Bf^+	Scleroderma	sd, Sd^+
Other skin structure variants			
Ear tufts	Et, et^+	Syndactyly	?
Sleepy-eye	se, Se^+	Breast blisters	*
Enlarged earlobes	?		

* Probably polygenic.

trait than were males. The gene symbol *sb* was assigned to this trait by Somes (1980).

Side sprigs. A frequent variant of single comb is the presence of side sprigs. These are regarded as a disqualification by the fancier and usually selected against. Side sprigs or lateral sprigs are extra spikes or points, one to three in number, which develop laterally from the posterior end of the single comb. They are usually classifiable in males by three to four weeks of age, but females must be several months of age before they can be classified accurately. Asmundson (1926) studied the inheritance of side sprigs in White Leghorns. His data clearly showed that this trait was not caused by a single gene. A better hypothesis was that two autosomal dominant complementary genes were producing the defect. This would also explain why the trait could occur in a high percentage of crossbred progeny from two stocks which might never have shown it themselves. Punnett (1923) and Jull (1930) have suggested a relationship between side sprigs and the crest gene (*Cr*). If such exists, then it would seem to imply that side sprigs as a phenotype may have more than one genetic basis.

Multiplex combs. Taylor (1946) studied a further group of variations of single comb which he collectively called multiplex combs. Comb types included triplex, quadruplex, and quintuplex. In five years of selection, his line of birds went from only 42.9 percent showing triplex combs with none more complex, to 32.5, 60.0 and 7.5 percent showing triplex, quadruplex, and quintuplex respectively. He concluded that it was possible to produce a pure breeding strain for this trait although his strain had not yet reached homozygosity. The multiplex comb had considerable variation in its phenotypic expression ranging from triplex to quintuplex with the greatest proportion (75 percent) being of the quadruplex type. In his initial stock and in subsequent F_1 and backcrosses, birds with side sprigs were quite prevalent. Taylor (1946) suggested that multiple factors were involved in multiplex comb inheritance, some of which had complementary action, and some were apparently identical with the side sprig genes reported by Asmundson (1926).

Rose comb (*R*). Rose comb is a breed characteristic of more than fifteen recognized breeds and varieties. This type of comb is broad, being nearly flat on top, covered with small regular papillae, and ending with a spike or leader at the rear (Figure 6.4). The length and width of the comb and carriage of the leader depend on the breed. For most breeds the leader projects horizontally back from the head or tilts slightly upwards. For the Belgium Bearded d'Anvers bantam, Rhode Island Red, and Wyandotte breeds, the entire comb curves to conform with the shape of the skull.

Bateson (1902) was the first to show that this type of comb was inherited as a completely dominant autosomal gene. The homozygous recessive type is single comb. This monofactoral inheritance was verified by several others (Hurst, 1905; Davenport, 1906; Bateson and Punnett, 1908), and the gene symbol R was assigned by Bateson and Punnett (1906). Abnormal segregation of the R and r alleles has been studied extensively by Mérat (1963), who suggested that a type of selective fertilization was involved with this locus.

Poor fertility is associated with R/R males in at least several breeds. Fertility of R/r^+ and r^+/r^+ males and of all three genotypes of females is perfectly normal. The extensive literature on this phenomenon is reviewed in Chapter 20. Sperm cells from R/R males have some fertilizing capability, but they cannot compete with sperm cells from R/r^+ males in multiple sire matings (Crawford, 1965). This can explain persistence of the single comb allele in rose-combed populations. Using field data, experimental matings, and mathematical simulation (Crawford, 1965; Wehrhahn and Crawford, 1965), it has been shown that about 16 percent single-combed progeny can be expected from rose-combed breeder flocks, even when single comb individuals are culled each generation.

There are several variations of rose combs, all of which probably have a genetic basis. Punnett (1923) suggested that the downward pointing rose comb of the Wyandotte was probably dominant over the Hamburg type. Somes (unpublished) found in an F_1 cross between Wyandottes and Rose Comb Bantams that the downward pointing rose comb was dominant. He also mated birds with bent spikes together and found that this defect did not breed true. About one quarter of the progeny had bent spikes, one quarter had ingrown spikes, and one half were normal. When ingrown-spiked birds were mated together, about 62 percent of the progeny had ingrown spikes while 38 percent had normal spikes. It would appear that these two defects are not inherited as simple recessives. There are two other rose comb variations that have been investigated.

Rugged and smooth rose comb (He^+, he^l). Surface characteristics of the rose comb have been shown by Cavalie and Mérat (1965) to be controlled by a pair of alleles at an autosomal locus. The dominant allele He^+ caused a rugged or rough surface. In day-old chicks the surface had a granular appearance, while in adults the comb is bulky with higher and more numerous spikes than that caused by the alternate allele. The recessive allele he^l produces a much smoother surface in both chicks and adults. These two phenotypes can be differentiated more easily in chicks than in adults. Penetrance for the he^l allele is complete in females, but not among males where modifying genes seem to have some influence. Both of these alleles also influence the number of spikes on single combs with homozygous smooth birds having fewer spikes than rugged birds (Cavalie and Mérat, 1967). Although it has not been investigated, these two alleles probably also influence the phenotype of walnut combs. For instance the smooth allele may well be responsible for the smooth-surfaced cushion comb.

Trifid rose comb. The tasselled and crested Watermaal breed is characterized by having a triple-spiked rose comb and a tassel crest with the middle spike being longer than the two outer ones. It is not known whether the Watermaal comb type has been studied, but other studies may give insight into the inheritance of this rose comb variant. The Silky rose comb type differs from other rose combs in that it is associated with a crest and is short in length with two or three points projecting from its posterior. Punnett (1923) suggested that shortening of the comb was probably due to the presence of the crest gene, while multiple points were probably due to another gene. However, others working with this trait (Cunningham, 1912; Bonhote, 1914; Dunn and Jull, 1927; Jull, 1930) showed that presence of the crest could account for both the shortening and the multiple points. The Watermaal rose comb is similar to that of the Silky breed and may also result from the interaction of crest and rose comb genes. However, Somes (unpublished) studied a triple-spiked rose comb phenotype in a noncrested strain of birds. When triple-spiked rose-combed birds were mated together, 72 single-spiked and 155 multiple-spiked birds were produced. When triple-spiked birds were mated to normal ones, 109 single-spiked and 91 multiple-spiked birds were produced. The latter were about equally divided between those with two and three spikes. These data would seem to indicate the action of a dominant gene as the causative agent for this type of multiple-spiked rose comb.

Pea comb (*P*). Pea comb is a breed characteristic of Brahma, Cornish, Araucana, Cubalaya, Sumatra, and several other breeds (Figure 6.4). It is sometimes called a triple comb. It is generally a low elliptical-shaped comb with three longitudinal rows of points running from front to back. The central row is the highest and most conspicuous. Pea comb inheritance is that of an incompletely dominant autosomal gene (Bateson, 1902). Partial dominance is not so noticeable when crossed with single-combed breeds with small wattles, but when crossed with Mediterranean breeds or breeds with large wattles, the heterozygous phenotype becomes one of a well-developed central blade with lateral ridges representing the two outer rows of points (Punnett, 1923). The gene symbol *P* has been assigned to this locus (Bateson and Punnett, 1906).

Munro and Kosin (1940) reported that pea-combed birds have a ridge of thickened skin that runs the length of the keel over the breast bone. This breast ridge is found only on birds carrying the *P* gene and is apparently a manifestation of it. Crawford (1961) verified this relationship and showed that a breast ridge was also characteristic of walnut-combed birds since they also possess the *P* gene. He found it to be useful in distinguishing between birds with rose and walnut combs, and between those with pea and single combs since classifying of comb types in young birds can be difficult.

Walnut comb (*R* + *P*). This comb type (Figure 6.4) results from the complementary interaction of the rose and pea comb genes (Bateson and Punnett, 1905b, 1906, 1908). Hence walnut-combed birds may have any of the following genotypes: *P/P R/R*, *P/p R/R*, *P/P R/r*, or *P/p R/r*. This comb type is characteristic of Chantecler, Kraienkoppe, Malay, and Orloff breeds. In the Chantecler breed, it is generally referred to as a 'cushion comb' and in the Malay breed as a 'strawberry comb', these names being descriptive of the slight conformational differences between them. The walnut comb is smaller than either the rose or pea comb. Generally a shallow transverse groove separates the posterior third of the comb from the anterior portion. In day-old chicks small hair-like feathers are present in the groove, while in older birds these are replaced by small feathers. Because of the small mass of this comb, it was incorporated into some breeds to make them less susceptible to freezing in cold weather (Wilfred, 1927; Petrov, 1935).

When working with pea and walnut-combed birds and various crosses, Petroff (1929) observed a significant deficiency of the pea comb phenotype from a few matings. Test crosses indicated to him that a dominant inhibitor gene was preventing the pea comb gene from expressing itself. He also found that this gene in two out of three single-combed Black Minora hens that he tested. This gene was apparently quite selective in interfering with the expression of the *P* gene but had no effect on the rose comb gene (*R*), so that genotypic pea-combed birds appeared to be single-combed, and genotypic walnut-combed birds appeared to be rose-combed. His observations could also be explained by chance deviations and classification errors since his numbers were small and four comb types were segregating in his matings. Further observation on present-day stocks is warranted.

Duplex comb (D^v, D^c). Several comb phenotypes are considered to be duplex. The Houdan has a 'leaf comb' which resembles a butterfly with its wings partially open; it rests in front of the crest. The Sicilian Buttercup has a 'cup' or 'buttercup' comb, consisting of a cup-shaped crown circled with medium-sized regular points and set on the centre of the skull (Figure 6.4). All other duplex combs, such as those characteristic of Crèvecoeur, LaFleche, Polish, Sultan, and some other breeds are known as 'V-shaped' or 'horn' combs, since two spikes project from the comb base just above the beak (Figure 6.4). The Breda has also been shown to have a duplex comb although in that breed only two small papillae are evident in the males (Bateson and Punnett, 1908). Large cavernous nostrils are characteristic of all duplex-combed breeds except the Buttercup.

From the work of Hurst (1905) with Houdans, Davenport (1906) with Polish and Houdans, and Bateson and Punnett (1905a, 1908) with Buttercups and Bredas, it is evident that all duplex combs are inherited as autosomal dominants. It was assumed that the same locus is responsible for each of the different phenotypes when Punnett (1923) assigned the gene symbol *D* to this locus. Davenport (1906) suggested that the large nostril feature is inherited as a recessive to normal nostrils, and Punnett (1923) suggested that comb type and possibly the occurrence of cerebral hernia are involved with this trait. Somes (1986) showed that the V-shaped and Buttercup combs are controlled by two different alleles at the same locus. He assigned the symbol D^v for the V-type allele and D^c for the cup allele. The D^v allele is incompletely dominant, and has a strong duplexing expression with complete penetrance, while the D^c allele is also incompletely dominant, but has a weak duplexing expression and 25 percent reduced penetrance in females. The D^v allele is dominant to the D^c allele. The wide cavernous nostrils are expressed with homozygosity for the D^v allele, but not with heterozygosity (D^v/d^+). The cup allele has wide nostril expression only in conjunction with the V-type allele (D^v/D^c). The leaf-type comb has not been studied relative to these two, but it is probably due to yet a third allele at this locus.

Breda comb (*bd*). The Dutch Breda is considered to have no comb; females are completely combless, and males show only two small papillae, one on each side of the centre line. Bateson and Punnett (1908) showed that lack of a comb was inherited as a single recessive trait and that its dominant allele, Bd^+, was necessary for the production of a comb. Punnett (1923) suggested the gene symbol *s* for this locus, but this was later changed to *bd* by Hutt (1949). Bateson and Punnett (1908) showed that the Breda carries the duplex gene (*D*), which accounts for the two papillae on the head of the male and the large

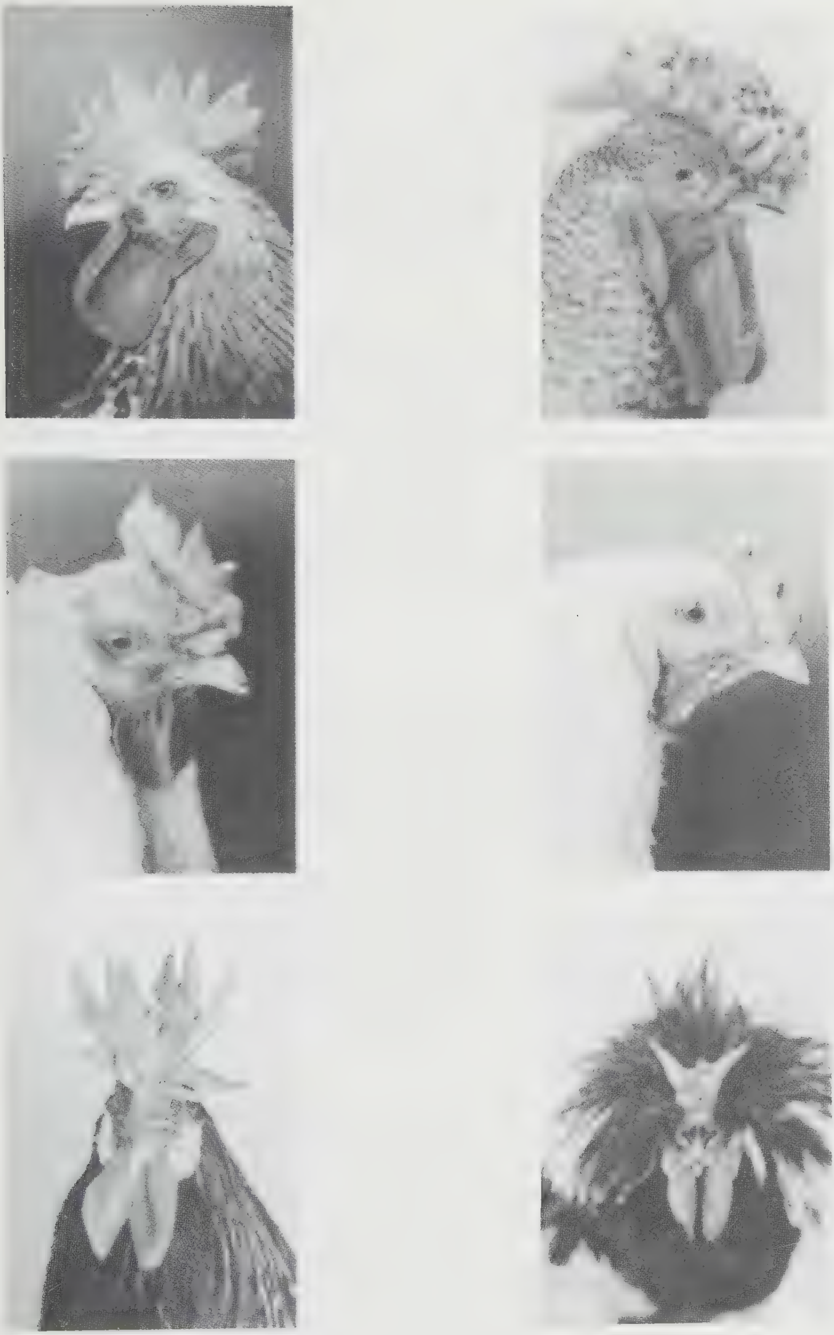

Figure 6.4. Comb variants. Upper left — single. Upper right — rose (R). Middle left — pea (P). Middle right — walnut ($R + P$). Lower left — Buttercup duplex (D^c). Lower right — V-shaped duplex (D^v). (Author's photos).

cavernous nostrils characteristic of this breed.

Variations In The Uropygial Gland

The uropygial or oil or preen gland is located on the dorsal surface of the tail overlying the free caudal vertebrae. It consists of a bilobed gland embedded in the skin with ducts leading from each lobe to a common papilla which projects vertically from the skin surface. The two duct openings are surrounded by small down feathers. Three genetic variants have been reported concerning this gland.

Uropygial (*U*). Hutt (1932, 1936b) reported briefly on a mutation which caused bifurcation of the papilla. He assigned the name uropygial and gave it the gene symbol *U*. Hutt (1949) gave more detail concerning it in his famous book. Uropygial was shown to be inherited as an incompletely dominant autosomal trait. Heterozygotes expressed the trait as a bifurcation of the papilla, with variation from heterozygotes not expressing it at all, to an almost imperceptible groove at the tip, to complete separation into two papillae joined at their base. Most homozygotes only showed two vestigial papillae at hatching and they lacked the entire uropygial gland, but a few homozygotes had functioning glands and double papillae similar to the heterozygotes. It was obvious that modifying factors were involved in gene expression.

Cleft and double uropygial papillae (*U-2*). Kessel (1945b) reported a second mutation that affected the uropygial gland. The affected birds all had functional oil glands, but some had double papillae while others had a definite cleft along the length of the papilla. In a cleft x cleft cross, observations were 14 double papillae, 21 cleft papilla, and 12 single papilla. From these data, a single incompletely dominant mode of inheritance was proposed. Because the homozygotes differed dramatically from the uropygial mutant reported by Hutt (1932), Kessel felt that this was a different mutant. However, it is also possible that Kessel's population was not segregating for modifying factors as was Hutt's, and that the same gene was involved. In that case, both homozygous and heterozygous phenotypes were being uniformly expressed at one of the less extreme levels characteristic of Hutt's birds. Hutt (1932) did have homozygotes and heterozygotes like those described by Kessel, but they were in the minority. A linkage test with the rose comb locus to which *U* is closely linked would have resolved the question. It is proposed that the gene symbol *U-2* be assigned to this trait.

Double oil gland papillae (*dgp*). A third mutant causing doubling of the uropygial gland papilla has been studied by Somes (1971). Expression of the trait ranged from completely double papillae to just a slight grove at the papilla tip. In this case, the trait segregated in crosses as a simple recessive. Mutants crossed with normals produced all normals, and when F_1 individuals were backcrossed to mutants both mutant and normal progeny were produced. Mutants mated together produced 382 progeny with all but seven being classified as mutant type. A linkage test carried out with the rose comb locus involving 623 birds gave independent segregation (51.5 percent crossover) verifying that this was not the *U* locus. The gene symbol *dgp* has been applied to this locus (Somes, 1980).

Variations In The Spur

Metatarsal spur development is characteristic of the male chicken, but not of the female. At one day of age, spur caps composed of hard keratin are present in both sexes. As the male grows older, spurs begin to develop out of the spur caps. At about six months of age, the bony spur fuses with the tarsometatarus and becomes permanently attached to the skeleton. Spurs continue to grow throughout the bird's life. There have been several mutants studied that involve spur development, and one of these, multiple spurs, is a standard breed characteristic.

Spurlessness (*sl*). This condition, which was reported by Kozelka (1933), prevented the development of spurs. The epidermal spur papillae were absent in day-old chicks, with an enlarged scale in their place. Spurlessness was frequently accompanied by an absence of scales along the inner surface of the shank. Some indication of sexual dimorphism was usually evident at maturity. Males developed slight protuberances at the spur sites, and these later developed into bony outgrowths from the shank and resembled deformed spurs, but the epidermal portion of the spurs was totally lacking. No such development was evident in females.

Genetic studies showed that spurlessness was inherited as an autosomal recessive and it was considered to be partially sex-limited since its effects were more evident in males than females. Kozelka (1933) assigned the gene symbol *s* for this trait, but it was later changed to *sl* (Hutt, 1949).

Auxiliary spur (*As*). Washburn and Smyth (1971) reported a mutant spur condition which they named auxiliary spur. This mutation was characterized as having extra bilateral spurs just above the normal ones. The extra spurs were small, not exceeding two cm in length by 18 months of age. They had normal attachment to the tarsometatarsus and continued to develop in length with age. Occasionally an even smaller third spur was present below the normal one. Auxiliary spurs could not be detected with accuracy at time of hatch. The best age to determine phenotype was 20-40 weeks.

Crosses with Buff Brahma bantams and Brown Leghorns showed that this trait was inherited as an autosomal dominant with complete penetrance. A partial dosage effect was suggested which related the third spur to homozygosity at this locus. The gene symbol *As* was assigned.

Double spur (*ds*). Domm (1931) reported double spurs in three Leghorn females following ovariotomy and speculated that this condition might be a mutation. Later, Warren (1946) studied the inheritance of a similar double spur condition. In chicks, double spurs showed as a prominent divided spur papilla. The dichotomy usually involved both legs, but sometimes only one was affected. In the adult, two spurs developed with the division extending throughout its length into the core. The two spurs on one leg sometimes differed in size with no fixed relationship to each other.

Warren's (1946) genetic studies indicated that inheritance of this trait was influenced by unknown factors. In general it behaved as an autosomal recessive, but females manifested it more often than males, penetrance was poor in homozygotes, and there was irregularity in dominance. The gene symbol *ds* is now associated with this trait (Somes, 1980).

Multiple spurs (*M*). The Sumatra breed is unique in having multiple spurs. Mature males may have three to five spurs on each shank. The second spur from the top is the longest with a shorter one above and one to three shorter ones below. Each spur has its own bony core, but only the longest ones are directly attached to the tarsometatarsus; the smaller ones are attached to a downward projecting flange-like protuberance. In newly hatched chicks, as well as in adult females, the normal single spur papilla is replaced by 3-5 enlarged and flattened scales. It is possible to classify this condition with over 98 percent accuracy at hatching.

Multiple spurs is inherited as an autosomal incompletely dominant trait (Hutt, 1941) with heterozygotes usually having two spurs and homozygotes having three to five. Some birds may also carry modifying genes that prevent the expression in heterozygotes. The gene symbol *M* has been assigned to this locus.

Molting spur. An unusual male was described by Kimball (1983) in which spurs were shed each year at the annual molt. The spurs never developed beyond short nubbins with rounded tips. They were wobbly and X-rays showed that the epithelial bony core never became united with the tarsometatarsus. Genetic studies were not made, but if this condition was inherited it was probably recessive since no F_1 or F_2 male progeny of this male were known to have the condition.

Spurs in females. Spurred females are not common among heavy chicken breeds, but they are found quite frequently among Mediterranean breeds. Bauer (1931) was unable to find any abnormalities in the ovaries or endocrine glands of spurred females. His spurred females reproduced and grew as well as did his nonspurred females. Others have also shown that spurred females perform well (Tandon and Iyer, 1954). However, Fairfull and Gowe (1986) found that spur length was negatively correlated phenotypically with egg production, egg weight, specific gravity, and Haugh units, and positively correlated genotypically with part-record egg production. Seasonal and dietary influences were not significant in influencing spur development in females, but the incidence of spur formation and spur length did increase with age (Christmas and Harms, 1982).

Goodale (1925) showed that spur development in females had a genetic basis, and by selection he developed a strain in which half of the females developed spurs. He suggested that inheritance was recessive. Tandon and Iyer (1954) also showed that this trait was inherited, but their crosses gave mixed and not very helpful results. Significant strain differences have been shown for this trait (Christmas and Harms, 1982; Fairfull and Gowe, 1986). Experience of the author (Somes, unpublished) and correspondence with an American Pit Game breeder whose females are spurred, indicate that when spurred females are crossed with light breeds (Leghorn, Polish, and Sebright) most of the female offspring develop spurs. Further genetics investigation is warranted.

Variations In The Skin

Four sclerodermal variants have been reported, each being inherited as an autosomal recessive. It is possible that some of these conditions may be caused by the same gene.

Dactylolysis (*dac*). Shoffner (1945) reported a peculiar sclerodermal condition of the feet of young chicks. Chicks were normal at hatching, but by one week of age their feet became hard, glassy, and slightly swollen. This was followed in a few days by cracks appearing in the epidermis of the soles. The cracks enlarged and became filled with blood exudate. The dermatitis was usually confined to the ventral surface of the feet, but in extreme cases it covered the whole foot. Distal portions of the toes atrophied and were sloughed. In some birds the condition was quite mild and they recovered in several weeks, but with severe cases recovery was prolonged to about 12 weeks of age.

Genetic matings showed that this condition was inherited and not attributable to disease or nutrient imbalance. Segregation data indicated that a single autosomal recessive factor was involved. It was later given the gene symbol *dac* (Somes, 1980). This trait was also considered to be semilethal because of the difficulty that chicks had coping with early life. In many ways this condition is similar to lesions seen with biotin or pantothenic acid deficiencies and somewhat similar to the blistered foot condition described by Somes (1970b).

Blistered foot lethal (*bf*). Somes (1970b) reported a down plumage abnormality which also caused lesions on the face and feet and was almost always lethal. The short down and lesions of feet and face appeared to be multiple manifestations of a single gene. A crippling syndrome in the same stock segregated independently. Fifty-eight chicks hatched and were classified as having short down only (25.8 percent), blistered feet only (8.6 percent), or both (65.6 percent). Chicks appeared healthy and lively at hatching, but 25 percent died during the first day, and 96 percent by 18 days of age. Only two birds lived to adulthood but they did not mature sexually. Blisters were present on the bottoms of the feet at hatching. Those living beyond eight days developed increasing dermatitis with subsequent necrosis and loss of toes, and they developed lesions in the corners of the mouth and eyes. The two that lived over a year continued to have cracked and bleeding feet, their beaks were elongated with the upper beak arched, and lesions developed in ears and mouth. The tail and flight feathers were very poorly developed.

The genetic basis of this complex trait was determined to be that of a single autosomal recessive. Segregation among progeny from matings of presumed heterozygotes was as expected, and where tested, the ratios of heterozygotes to noncarriers were also as expected. The gene symbol *bf* was eventually given to this trait (Somes, 1980).

Avian ichthyosis (*dehy*). Avian ichthyosis was characterized by epidermal hyperkeratinization of the majority of the integument and its specialized derivatives, although there was regional variation in its effects (Abbott and Sawyer, 1974; Sawyer and Abbott, 1974). At hatching, chicks had wiry down and their backs had patches of thickened white scaly skin. The epidermal layer was about four times the normal thickness. By one week of age scaliness had spread to the shanks, vent, beak, and eyes. Severe crippling followed as digits were lost. The condition persisted into adulthood with mature birds being covered with thickened scaly areas. Only about 40 percent of affected birds lived. At maturity, they were about 75 percent of the size of normal birds and were about seven weeks late in reaching sexual maturity (Abbott and Sawyer, 1974). Developmental studies showed that the first histological changes appeared in 19-day embryos, and that mitotic activity of affected skin was two to three times greater than normal controls at one day of age (Renden and Abbott, 1980).

Avian ichthyosis is inherited as an autosomal recessive trait. It was derived from a stock carrying the 'dehydrated' trait (Taylor and Stinnett, 1957), which was originally completely lethal. The gene symbol *dehy* has been applied to the ichthyosis-dehydrated syndrome (Somes, 1980).

Scleroderma (*sd*). Scleroderma (progressive systemic sclerosis) is an autoimmune disease characterized by fibrosis of skin and internal organs. The scleroderma mutation in the chicken is an excellent animal model for the study of human scleroderma. This chicken trait was first reported as a phenodeviant called 'self-dubbing' (Bernier, 1961). The comb of chicks at 2-3 weeks of age would hypertrophy and then undergo necrosis, with the end result being the same as dubbing. Genetic studies indicated that this condition had a recessive inheritance but probably with polygenes being involved. Subsequently the mutant stock was studied more fully and its resemblance to scleroderma was investigated (Gershwin et al., 1981). Besides the early progressive involution of the comb, dermal fibrosis and distal polyarthritis also occurred within the first six weeks of life. Approximately 40 percent of affected birds die by four months of age due to secondary cutaneous infections of the comb and digits. Birds that lived longer developed fibrosis of the esophagus and mononuclear infiltration of the heart and kidney, with prominent occlusion of small medium-sized blood vessels. These birds also developed rheumatoid factors, antinuclear antibodies, and antibodies to collagen. An excellent summary of all the features of this trait and references to the investigations of some of these was prepared by Van der Water and Gershwin (1985).

Genetic studies showed that the condition was not evident in F_1 progeny, and about half of the backcross progeny were affected, but there was only about four percent incidence in the F_2 generation. An autosomal recessive was suggested as the casual agent along with modifying genes (Gershwin et al., 1981). The gene symbol *sd* has subsequently been assigned to this trait (Somes, 1984).

Other Skin Structure Variants

Several other skin variants are known. Three of them affect the head region, one affects the feet, and another causes skin defects over the breast.

Ear tufts (*Et*). The South American Araucana is unique in several respects. It is the only breed that has the unusual ear tuft trait. Ear tufts are feather-covered epidermal appendages that project from the sides of the head in the vicinity of the ear opening. Expression of this trait varies widely, being bilateral as well as unilateral, and having variation in size, direction of peduncle projection, and location of attachment (Figure 6.5). Expression of the ear tuft gene includes an effect on structural rearrangement of the ear. The external opening is quite irregular in shape and the external auditory canal is either reduced in length or completely absent (Somes, 1978b). Studies of the embryonic development of ear tuft and ear canal suggest that the structural abnormalities are derived from incomplete fusion of the hyoid and mandibular arches over an area from the distal part of the ear opening to the neck (Pabilonia and Somes, 1981, 1983).

The Araucana ear tuft trait is inherited as an autosomal dominant trait that is lethal in homozygotes. It has been assigned the gene symbol *Et*. Prenatal death is also increased in heterozygotes varying from 20-42 percent, and penetrance is reduced by 4-14 percent. Posthatch mortality is significantly greater among tufted chicks than nontufted ones (Somes, 1978b; Somes and Pabilonia, 1981). Occasionally an *Et/Et* bird will escape the lethal effects of this gene. Such a bird was reported by Somes and Pabilonia (1981), but this male was unable to pass his 'escaper' ability to the next generation.

Sleepy-eye (*se*). A rather unusual trait appeared in five of 1233 birds of a randombred population (Somes, 1968). The 'eyes-half-closed' classification gradually increased in its

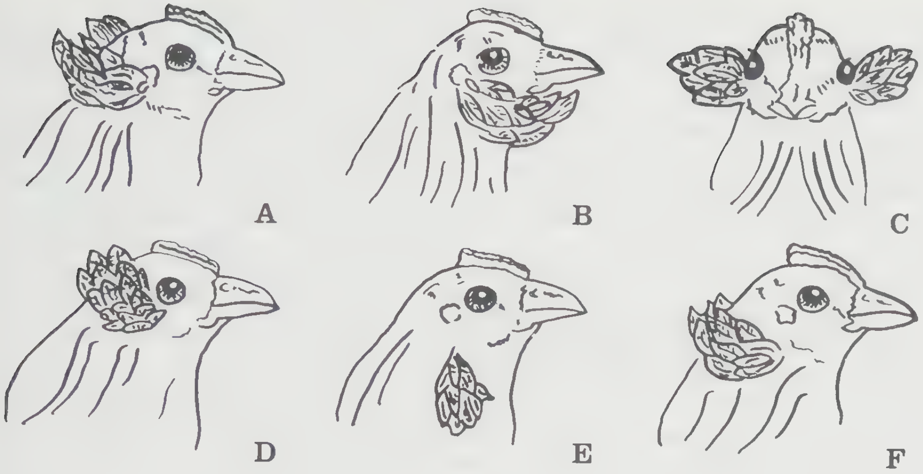

Figure 6.5. Types of ear tufts (*Et*) A and F — back swept. B — front swept. C — horizontal. D — vertical. E — teardrop. (From Somes, J. Hered. 69:94, copyright 1978 Am. Genet. Assoc.).

frequency and eventually it became known as sleepy-eye (Figure 6.6). It affected the function of the lower eyelids. Chicks were hatched with their lower eyelids being partly to fully closed. When startled they could open their eyes fully. This condition remained with the birds throughout life. Although not a lethal in itself, sleepy-eye was semilethal since in extreme cases it interfered with the chicks' ability to find food and water. In order to see better, birds would tip their heads forward and look over their raised eyelids.

Genetic studies showed that the sleepy-eye trait was inherited as an autosomal recessive with complete penetrance. It was assigned the gene symbol *se*.

Enlarged earlobes. An unusual trait that has not been investigated genetically is the greatly enlarged earlobes that are characteristic of the White-Faced Black Spanish breed. It seems obvious that this condition has a genetic basis, but the nature of it is still unknown. Where most breeds have compact well-defined earlobes below the ears, the White-Faced Black Spanish earlobes extend well below the wattles, meeting in front and going well back on each side of the neck. The trait is not fully expressed until the birds are two years of age, which may be one reason why it has not been studied.

Syndactyly. Syndactyly is a condition in which the third and fourth toes are fused, usually by continuous webbing between the toes (Figure 6.6). The extent of webbing varies distally and between toes. In extreme cases the toes are tightly tied together, and the nail of the fourth toe may be bent out of its median position making walking difficult. The condition is usually expressed in both feet. Syndactyly has been associated with both ptilopody and brachydactyly by several workers (Davenport, 1909; Danforth, 1919a, 1919b; Jaap, 1939). They proposed that the same gene was responsible for all three of these conditions and that modifying genes and genetic background determine the phenotype of each individual. Davenport's (1909) data showed syndactyly to be dominant. This would be expected if one of the dominant shank feathering genes were responsible for it. Danforth (1929) suggested that both of the dominant shank feathering genes were able to produce syndactyly. A relationship between these two traits was further strengthened with the appearance of the single gene multitrait mutant *psp* reported by McGibbon and Shackelford (1972) and Smyth (1981) in which syndactyly and ptilopody were shown to be caused by the same recessive gene.

There probably is more than one genetic type of syndactyly. Two studies have been made of this trait in clean-legged stock, where ptilopody genes could not be involved. One study used White Plymouth Rocks (Warren, 1950) and the other one involved junglefowl stock (Hollander and Brumbaugh, 1969). In both cases, results from crosses were difficult to interpret. Warren (1950) found that the normal parent had a major influence on F_1 progeny; he got only 1.8 percent syndactylism in one group of F_1 crosses but 41 percent in another, syndactyl x syndactyl crosses produced 62 percent affected progeny, and there were only 11.9 percent affected in the F_2 generation. The comparable results from Hollander and Brumbaugh (1969) were zero, 40 and one percent affected birds. Although Warren (1950) had somewhat more affected individuals, these two groups may well have been working with the same genetic complex. Hollander and Brumbaugh (1969) proposed complementary gene action of a dominant and a recessive factor along with variable penetrance and expressivity. Warren (1950) did not suggest a mode of inheritance, but said that there was no evidence of sex-linkage.

Breast blisters. Breast blister development in broilers is common, and differences in incidence between breeding groups are influenced by body weight, sex, and breast feathering (Gyles et al., 1957). Genetic factors are also involved in the development of blisters as well as in breast feather coverage. Gyles et al. (1959) selected divergent lines, one susceptible and the other resistant to breast blister development. Estimates of heritability calculated from the sire, dam, and combined components for the susceptible line were .16, .24, and .20 for blister condition, and .50, .38, and .44 for breast feather coverage. In the resistant line, heritability was zero for blistering, and it was .02, zero and .01 for breast feather coverage. It is apparent that inheritance of breast blister incidence is polygenic.

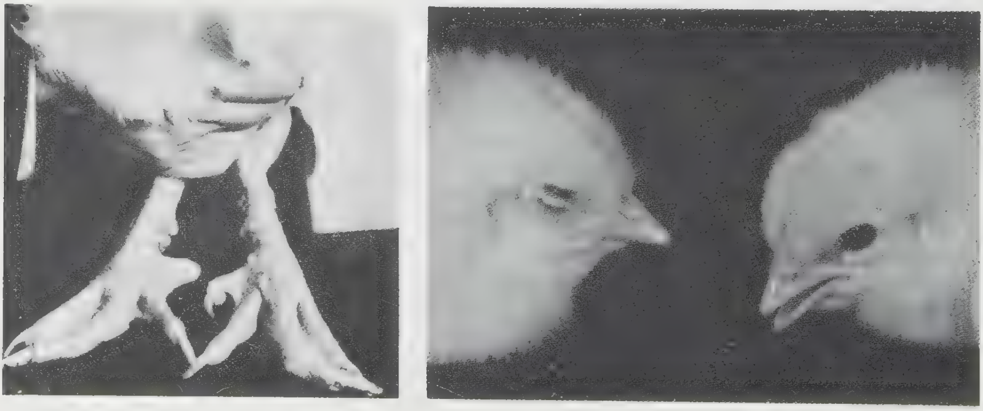

Figure 6.6. Skin structure variants. Left — syndactyly (from Warren, J. Hered. 41:32, 1950). Right — sleepy-eye (*se*) and normal chicks (from Somes, J. Hered. 59:375, copyright 1968 Am. Genet. Assoc.).

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Chapter 7. MUTATIONS AND MAJOR VARIANTS OF MUSCLES AND SKELETON IN CHICKENS

Ralph G. Somes, Jr.

In this chapter the mutations which affect muscles and the skeleton will be described. They will be presented in four sections as genetic variations in muscles, in the axial skeleton, in the appendicular skeleton, and in body size. Many of them are at least partially lethal to affected individuals. Those that are completely lethal will be described in Chapter 11.

MUSCLE MUTATIONS

Mutations of the musculature are not very common in the chicken. Only three such mutations are described here.

Hereditary ventricular septal defects. Ventricular septal defects are among the most common congenital cardiac abnormalities in animals. A defect of this kind has been reported for the domestic fowl by Siller (1958). Ventricular septal defects consisted of openings in the septum, with considerable variation in their conformation and size of the openings between the left and right ventricles. Functional valves developed many defects which prevented significant shunting of blood, and it appeared that in older birds many septal defects actually closed. Rychter et al. (1960) and Rychter and Lemez (1978) studied the morphogenesis and development of this condition in embryos, and Siller (1958) studied it in hatched chicks. Only young birds with the most severe defects showed clinical symptoms. All others appeared to be healthy, and as adults they were able to reproduce. However, birds with septal defects were less able to withstand extremely cold weather.

A complex inherited basis was proposed for this condition. In the six Brown Leghorn inbred lines where it was studied, the frequency of defects ranged from 31.4-84.2 percent in the three high incidence lines, and from 3.6-5.9 percent in the three low incidence lines. Crosses of low x low, high x high, and low x high incidence lines produced 0, 10.7, and 1.1 percent defects in the progeny. It would appear that either this condition was inherited as a recessive with very low penetrance, or multiple factors were involved.

Muscular dystrophy (*am*). A particular stock of New Hampshire chickens was obtained in 1953 for study at the University of California-Davis. These birds had been previously selected for large breast muscle development and were unable to raise their wings. This condition was described as a muscular dystrophy by Asmundson and Julian (1956). In that first report, it was reported as being inherited as an autosomal recessive to which the gene symbol *am* was applied. However, heterozygotes have wider breast muscles than do normals (Asmundson and Julian, 1956), along with the histopathological changes and microscopic lesions characteristic of homozygous dystrophics (Julian and Asmundson, 1963; Julian, 1979; Farrell and Tureen, 1968). Some consider the trait to be a codominant, but full manifestations of the disease as measured by the 'flip test' (the inability of the bird to turn over when placed on its back) are expressed as an autosomal recessive (Julian, 1979) (Figure 7.1). This condition is actually a polygenic trait with the *am* gene having a major effect, because it was possible to produce four separate dystrophic lines with distinctly different characteristics; these were early and late onset lines and high and low fat infiltration lines (Holliday et al., 1968).

This particular genetic trait of chickens has been used in more biomedical studies than any other. It is now considered to be an appropriate animal model for investigations of hereditary muscular dystrophy. There is a very large volume of research literature, including several general reviews (Julian and Asmundson, 1963; Asmundson et al., 1966; Julian, 1973; Julian, 1979), and reviews of special aspects such as muscle properties (Randall and Wilson, 1980), enzyme studies (Cardinet et al., 1972), physiological and histochemical characteristics (Wilson et al., 1979), oxidative metabolism (Ashmore and Doerr, 1970), biochemical studies (Owens, 1979), histopathological studies (McMurtry et al., 1972), and electrophysiological observations (Warnick et al., 1979).

Briefly, the pathology of this dystrophy consists of gross hypertrophy of muscles followed by atrophy in adulthood. 'White' muscles are affected before the 'dark' muscles. Interference with the action of the supracoracoideus results in stiffening or locking of the wing joints. Microscopic changes consist of an increase in nuclei number, fibre size variations, fibre splitting, destruction of fibres, and presence of ringed fibres. Infiltration of fat occurs at the sites of muscle fibre loss.

Cornish muscular dystrophy. A muscular dystrophy syndrome quite similar to the one reported by Asmundson and Julian (1956) was investigated in a Cornish population (Wagner et al., 1970). The superficial parts of the supracoracoideus, pectoralis, and coracobrachialis muscles were the most sensitive to this syndrome; they became an opaque yellowish color. Muscle weight varied considerably with some muscles becoming hypertrophied while others were atrophied. Histologically, affected muscles showed the characteristic degenerative changes of muscular dystrophy; these included fibre degeneration, fibre splitting, necrosis, fat infiltration, vacuolar degeneration, and an increase in connective tissue. Using wing stiffness as a measure of onset of the syndrome, some progeny could be classified as early as four weeks of age with the most severe signs having occurred by 12

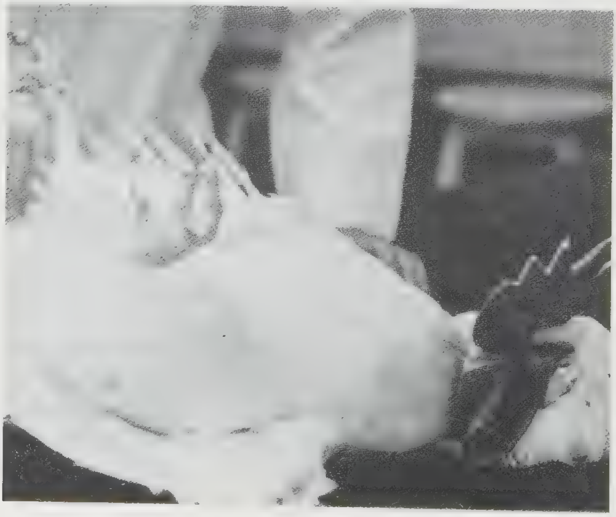

Figure 7.1. Muscular dystrophic homozygote (*am*/*am*), unable to right itself during a 'flip test'. (Author's photo).

weeks of age. Measurements of serum creatine phosphokinase activity in muscular dystrophic Cornish showed it to be significantly elevated compared to nonaffected Cornish at six through 32 weeks of age and it remained high, whereas in the New Hampshire muscular dystrophy syndrome it decreased at a variable rate as the birds aged to 25 weeks of age (Wagner et al., 1971).

The inheritance of this trait was studied using three types of crosses. Nonaffected x nonaffected produced no affected chicks. Affected males x nonaffected females produced 30 percent affected chicks, while affected x affected produced 63 percent affected chicks. Nonaffected parents were randomly selected from a population of 400 birds with no knowledge as to their genotype relative to this trait, and so the first two types of crosses were not very meaningful unless pair matings were made which apparently was not the case. The third cross indicated that this condition in the Cornish breed did not seem to be inherited as a single autosomal recessive; either it was inherited as a recessive with reduced penetrance, or it was polygenic. Hence it would appear to be a different condition than the *am* muscular dystrophy. Julian (1979) stated that the original Cornish stock from which this dystrophy came was in fact a mixture of other breeds, one of which was the original dystrophic line of New Hampshires. It may be that the Cornish condition was caused by the *am* gene, and that the difference between the two types is due to the special genotype of the Cornish breed interacting with the dystrophy gene. However, in this type of dystrophy the heterozygotes were indistinguishable from nondystrophics (Wagner et al., 1970).

VARIANTS OF THE AXIAL SKELETON

Inherited variations of the axial skeletion include mutations affecting the beak, head, neck, and body. In this section they will be covered in this anterior-posterior order. They are summarized in Table 7.1.

Cerebral hernia. Cerebral hernia is frequently associated with a crest, particularly in those birds with a very large crest. This condition was first observed by Tegetmeier (1856). Krautwald (1910) showed that the underlying cause of the hernia was an accumulation of fluid in the third and fourth ventricles of the brain thus causing an upthrusting of the cerebral hemispheres and the overlying frontal bones. The relationship between this abnormality and crest formation, with its associated elongation and erection of feathers, is not known. There does appear to be a direct relationship between the size of the crest and the extent of the cranial abnormality. Tasselled breeds do not show the condition. According to Jeffrey (1977) the Brabanter and Watermaal breeds should not show a protuberance of the skull, and it is further considered a disqualification for the Watermaal breed; all of these are tasselled breeds.

The genetic basis of cerebral hernia is still not fully understood. Cerebral hernias have always been recognized as being closely associated with the head crest. However a number of early workers considered that it was nevertheless caused by a separate recessive gene (Davenport, 1906; Dunn and Jull, 1927; Dunn and Landauer, 1930). Fisher (1934), and Warren and Hutt (1936) showed that this was not the case, and that both traits are manifestations of the *Cr* gene, each being separately affected by modifying factors and thus leading to considerable variation. Dunn and Landauer (1930) showed that cerebral hernia was linked to the dominant white locus by about 11.5 crossover units, and Warren and Hutt (1936) found that the crest locus was also linked to the dominant white locus by about the same amount (12.5 units). More recently, Requate (1960) studying the Dutch White-

Table 7.1. Listing of inherited variations in the axial skeleton of chickens.

Trait and gene symbol		Trait and gene symbol	
Cerebral hernia		Crooked neck	crn, Crn^+
Hereditary exencephaly		Crooked keel	
Sparrow head	sph, Sph^+	Supernumerary ribs	
Snub nose	sno, Sno^+	Number of vertebrae	
Crossed beak		Kyphoscoliosis	
Short mandible	sm, Sm^+	Spondylolisthesis	
Short upper beak	su, Su^+	Dominant rumplessness	Rp, rp^+
Cleft palate		Recessive rumplessness	rp-2, Rp-2^+
Palatal pits	pt, Pt^+		

Crested Dwarf fowl concluded that 'skull tuberosity' was not due to the crest gene, but was due to a separate dominant gene Kf_H, which was very closely linked to the crest locus. From a backcross mating of 86 progeny, he found linkage distance to be 3.5 map units.

One aspect of cerebral hernia development that does not seem to have been addressed in any of the published studies is the possibility of an interaction between comb type and expression of cerebral hernia. Hernias in conjunction with large crests are always associated with the very small V-type duplex comb. Birds heterozygous for crest generally do not have cerebral hernias, although Warren and Hutt (1936) reported a few. These F_1 birds have much larger comb masses since they usually have resulted from crosses with breeds of other comb types. Crested single and crested rose comb birds do not have cerebral hernias. It may be that this trait can only be expressed when the top of the head is more or less free of a comb mass. A cross between a crested Polish and a noncrested La Flèche with a V-shaped duplex comb might provide an answer to this question. If there really is an interaction with comb mass, then this trait would appear to be inherited as a recessive when outcrossed to breeds not having the V-shaped duplex comb.

Hereditary exencephaly. An unusual trait in which chicks showed varying degrees of exencephaly was reported by Mellen (1959). Expression of the condition ranged from scarcely noticeable hydrencephalocele to massive herniation. The basic defect was cranioschisis formation usually in the frontoparietal region with extrusion of brain parts being a secondary manifestation. This condition had no adverse prenatal effects, but usually proved lethal within a few days after hatch due to mechanical injury and cannibalism. Males and females were affected equally. With special effort, exencephalic birds could be raised to maturity. Even as adults, most of the cranioschisis remained, although a few closed over.

Genetic studies over a five year period showed that exencephaly had recessive inheritance, but its exact mode of inheritance was not apparent from the data obtained. Reciprocal matings between affected birds and unrelated normal birds produced all normal chicks. However, affected x affected produced 13.6 percent affected, while normal carrier x normal carrier produced 8.9 percent affected. If this trait was caused by a single recessive gene, then its penetrance was quite low. Polygenic inheritance may well be the mode involved.

Sparrow head (*sph*). A delayed lethal condition which had an effect on skeletal development was reported by Gyles et al. (1956). Birds with this condition were normal at hatching but developed abnormally from nine weeks of age to maturity. There was a progressive shortening of the head bones and beak, thus leading to the 'sparrow head' designation. In some cases, leg bones were poorly formed and crooked. All affected birds died between eight and 15 months of age with none reaching sexual maturity. Inheritance was deemed to be recessive since six of 30 progeny from a pair mating showed the trait. Six normal appearing full sibs from this mating produced 18 of 49 chicks with the condition. It is proposed that the gene symbol *sph* be assigned to this trait.

Snub nose (*sno*). Smyth (1978), as cited by Somes (1978), studied an autosomal recessive trait which showed varied expression, but whose primary characteristic was a short broad head with a shortened beak. Varying degrees of parrot beak were present in a high proportion of the mutants. Extreme cases showed crossed beaks and reduction in body size presumably due to difficulty in eating. These birds were not good reproducers. The gene symbol *sno* was assigned to the trait.

Crossed beak. A wide variety of poultry breeds can exhibit the crossed beak trait. Occasionally a similar trait will also appear associated with unilateral microphthalmia or anophthalmia, but this type is not genetic and is caused by abnormal development of the eye. Both of these types of crossed beak are greatly influenced by incubation conditions (Landauer, 1937). The most common expression is that in which chicks hatch with normal beaks, and crossing of the beak begins to be evident at 1-2 months of age, with maximum expression developing by 3-4 months of age. The abnormality may affect either the upper or lower beak, but the upper beak is much more often the affected part. It will develop a lateral bend, more often to the right, which may vary from only the tip being affected to the whole upper beak being affected. The visceral and cranial skull also show definite asymmetry which was considered to be the basic cause of the beak deformities (Landauer, 1938). Mercier and Poisson (1927) found that the tongue of birds with crossed beak was asymmetrical in structure and developed a horny tip with excessive cornified tissue on its surface.

The genetic basis of the crossed beak trait has been studied by Mercier and Poisson (1925), Taibell (1929), and Morris (1932, 1934). They all stated that it was inherited, but did not commit themselves on its mode of inheritance. Hutt (1932) suggested, based on his own data, that it was probably inherited as a simple recessive. Extensive studies by Landauer (1938) showed that the trait was recessive, but no true-breeding individuals were obtained even after considerable inbreeding, and many birds known to be genetically cross-beaked did not even show the trait. Mercier and Poisson (1925) had suggested that some of their heterozygous stock had also shown the trait. Landauer (1938) considered the inheritance of this trait to be recessive, but complex. He was further able to demonstrate breed differences with experiments in which he induced crossed beaks by using ethyl carbamate injections (Landauer, 1956).

Another type of crossed beak was studied by Landauer (1938), in which chicks exhibited the defect at hatching. He found this congenital type only in White Leghorns and crossbred birds of Leghorn ancestry. It differed from other types only in that some chicks with crossed beak became normal as they advanced in age. Crosses between this congenital type of crossed beak and the late onset type produced only normal birds (Landauer, 1938). When birds with congenital crossed beak were mated *inter se* they produced a higher proportion of affected offspring than did the late onset type. A recessive multifactoral basis with reduced penetrance was proposed for this trait.

Short mandible (*sm*). A condition in which the lower mandible was shortened to about half the length of the upper beak was reported in White Leghorns by McGibbon (1946). In some cases the rami in the midregion would buckle up or down, and when bowed up would result in a right-angle bending of the shortened mandible; this situation would cause the mouth to be permanently propped open. Extreme buckling was almost always associated with a rolling under of the tip of the tongue. Long bones of the appendages were not affected. About 50 percent of affected embryos hatched, but few chicks survived because of inability to eat or drink. Those that did survive had a lower mandible with nearly normal length at 5-6 months of age, but because of a persistent downward curving of the upper beak, it had to be trimmed frequently.

A series of matings using carrier, normal, and affected birds showed that the trait was inherited as a single autosomal recessive. In a 'short lower' x 'short lower' mating, two normal and 37 affected chicks were produced, indicating variable expressivity. Hutt (1949) assigned the gene symbol *sm* to this semilethal autosomal trait.

Short upper beak (*su*). This mutation, which was reported by Landauer (1941), affected the upper beak as well as the long bones of the appendages. The wing bones were less affected than the leg bones. Shortening of leg bones followed a proximodistal gradient with the tarsometatarsus being most affected. The upper beak was shortened by amounts varying from one mm to half its total length. There was no correlation between degree of beak shortening and degree of limb shortening. About 13 percent of affected embryos hatched. Early posthatch mortality was high due to eating difficulties. Of those surviving to maturity, the beak remained short in some while it developed to a normal length in others; some developed a crossed beak with the upper beak curvature being more frequently to the right.

Genetic analysis of this condition showed that it resulted from the action of a single autosomal recessive gene. Although the trait could be detected as early as the ninth day of incubation, its lethal action was not evident prior to the 18th day. Landauer (1946) selected against the lethal action of this gene for five years and in so doing was able to increase the hatchability of homozygotes to 77 percent. He was also able to raise the percent with normal beaks from zero to 71 percent. The gene symbol *su* was assigned to this locus by Hutt (1949).

Cleft palate. A rather interesting and genetically complex trait, cleft palate (CP), was studied by Roberts et al. (1974) and Juriloff and Roberts (1975). Palates of affected chicks were either unilaterally or bilaterally clefted. Expression ranged from mild to severe with the most severe expression being partly attributable to the semilethality associated with this trait (Juriloff and Roberts, 1975). The Minnesota 420 inbred White Leghorn line was the initial source of the CP trait; it had an average incidence of about 17 percent. The base population showed very limited variation when on a normal breeder diet, but subjecting the females to a stressful riboflavin-deficient diet increased the variation sufficiently to allow for selection and interpretation of the genetic basis involved. They were able to increase the incidence of CP to 29.7 percent in one year and to 47.4 percent in the next (Roberts et al., 1974).

Crosses between the CP line and a New Hampshire line were observed under both normal and riboflavin-deficient maternal nutrition. No indication of sex-linkage or of differences in sex expression were observed. The data best fitted a model of three recessive loci during normal maternal nutrition, and two recessive loci during maternal deficiency. Additive penetrance was probably due to three or four minor loci. Penetrance was 50-75 percent. The difference in major loci involved relative to diet was suggested to be due to one locus which when homozygous normally mimicked some aspect of the riboflavin deficiency, and thus it became irrelevant when a deficiency was actually imposed (Juriloff and Roberts, 1975). The selection progress made by Roberts et al. (1974) would seem to suggest that the recessive alleles at the major loci involved were fixed and that the increased incidence of CP progeny from one generation to the next was due to elimination of additive penetrance-controlling genes.

Palatal pits (*pt*). Palatal pits, small pits that were present either unilaterally or bilaterally on the palate, were discovered by Juriloff and Roberts (1977) at very low incidence (0.79 percent) in the Minnesota 420 White Leghorn inbred line. This was the same stock in which the cleft palate trait was segregating, but there was no evidence that the two traits were related. Presence or absence of palatal pits was recorded in the F_1, F_2 and backcrosses involving the cleft palate trait. No palatal pits were evident in either the F_1 or F_2 generations, but 0.88 percent were observed in the backcrosses.

A simple recessive mode of inheritance was proposed based on the observed gene frequency of 0.09 in the Minnesota 420 line, absence of pits in the New Hampshire line used in the crosses, and the assumption that frequency of pits did not change in the Minnesota 420 line. The expected numbers of chicks with palatal pits in the backcross generation were actually found. The gene symbol p^l was assigned to this trait. However, the symbol *p* has already been assigned to the pea comb locus and so Somes (1980) proposed using the gene symbol *pt* instead.

Crooked neck (*crn*). The crooked neck trait was discovered in Brown Leghorns (Jull and Quinn, 1931a). Chicks appeared normal at first, but by the time they were half grown they had developed a crooked neck which was usually twisted to the right. Both sexes were affected. As birds grew older, the condition usually worsened such that they were eventually unable to eat and drink. Females laid very few eggs and males were unable to mate. It was suggested that the etiology of the crooked neck condition was due to cessation in growth or misplacement of one or more vertebral ligaments.

Matings between four carrier males and 14 carrier females produced 129 normal progeny and 27 crooked neck progeny. Although the observed number of crooked neck progeny was about 30 percent lower than would be expected, Jull and Quinn (1931a) concluded that this trait was inherited as a single gene. The gene symbol *crn* has been assigned to this locus (Somes, 1980).

Crooked keel. The keel on the sternum provides attachment for muscles of the breast. Although the keel bone is usually straight, it can be bent and it may show varying degrees of concavity. Bending to the right is more common than bending to the left (Warren 1937). Various factors such as sex, age at roosting, width of roosts (Platt, 1932, 1933; Warren, 1937; Carstens et al., 1936), and genetic susceptibility (Warren, 1937) have all been shown to be involved in manifestation of this trait.

Biegert (1937) found that individual selection against crooked keel was ineffective because of the strong environmental influences on its expression. However, Warren (1937) was able to demonstrate a genetic influence by using family selection. Crooked-keeled parents from high incidence families produced progeny of which 80 percent were crooked-keeled, while straight-keeled parents from low incidence families produced progeny of which only four percent were affected. Six generations of additional selection only reduced incidence in the low line to three percent and raised incidence in the high line to 88 percent. Reciprocal crosses gave intermediate incidence and no indication of sex-linked genes being involved. A multifactoral mode of inheritance was proposed by Warren (1937) for susceptibility to keel bending. The actual expression of the trait depended to a large extent on environmental factors. For example, Warren (1937) showed that in genetically susceptible birds, the frequency of crooked keels could be lowered from 92 percent to 11 percent at 24 weeks of age by providing 2.5 cm roosts at 12 weeks of age instead of at four weeks of age. When nonsusceptible males were raised with roosting conditions conducive to maximum development of crooked keels, only three percent developed them.

Knize et al. (1983) studied the incidence of crooked keels in three inbred lines over a three year period using F_1 crosses and backcrosses. Their C and Iowa lines never showed crooked keels, but the W line had an incidence that ranged from 0-62.5 percent depending on the year. F_1 crosses between Iowa x C, Iowa x W, and W x C produced 0, 44.4-100, and 33.3-100 percent incidence of crooked keels respectively. When the Iowa x W F_1 was backcrossed to the Iowa line the incidence was 40 percent, and when backcrossed to the W line it was 64 percent. These data appear to indicate that crooked keel is dominantly inherited, and that genetic susceptibility and adverse environment interact to produce the trait. Warren's (1937) work showed that the abnormality resulted from a specific weakness in the sternum, and not from faulty ossification.

Supernumerary ribs. The chicken normally has seven pairs of ribs. The first two are floating, while the next four articulate with the sternum. The seventh rib does not terminate with the sternum, but is instead a 'false rib'. Herrick and Herrick (1956) described the presence of supernumerary eighth ribs in White Leghorns and Leghorn-heavy breed crosses. These extra ribs were false ribs somewhat like the seventh pair.

Pryszcz (1987) studied the inheritance of the supernumerary eighth pair of ribs in Partridge Greenleg chickens. Offspring from father-daughter matings of 8 pair x 8 pair, 8 pair x 7 pair, 7 pair x 8 pair, and 7 pair x 7 pair birds had a incidence of 94.3, 45.3, 1.1, and 3.1 percent with eight pairs of ribs respectively. A consistent sex difference was evident, with more males than females having additional ribs. A polygenic mode of inheritance was proposed with possibly one major gene and several modifying genes some of which may be sex-linked.

Higher blood serum alkaline phosphatase activity at one day of age and lower Ca content of bones in birds with extra ribs indicated a slower ossification of the skeleton of these birds (Zieba et al., 1985). A decrease with age in the numbers of individuals with extra ribs suggested that this trait caused higher mortality both embryonically and postembryonically (Pryszcz, 1987) and at later ages it weakened fecundity and fertility (Zieba et al., 1983).

Number of dorsosacral vertebrae. Promptoff (1929) studied variations in the structure and numbers of dorsosacral vertebrae. Normally, chickens have 14 ribless cervical vertebrae and then seven with ribs attached, ending with Vertebra 21 which marks the beginning of the dorsosacral region of the pelvis. Normally there are four dorsosacral vertebrae (V21-V24) each characterized by well developed transverse outgrowth processes. Vertebra 25 in a normal pelvis begins the lumbrosacral region; it has an entirely different structure.

Promptoff (1929) identified three main types of dorsosacral vertebrae using data from dissection of about 4000 chickens. The normal type consisting of four dorsosacral vertebrae was found in about 75 percent of all the material examined and was widespread in all breeds. The bantam type had only three dorsosacral vertebrae (V21-V23) with no transverse processes on V24. This type and the normal type were common among bantams chickens. In the bantam type, the acetabulum was moved forward to about the level of V28. The lengthened type had five vertebrae (V21-V25). It was found in about 25 percent of the material and was associated with the larger breeds. In this type V25 had the same broad transverse processes as V24.

Genetic studies of a trait of this type would be difficult to carry out. Matings must be made without knowing the parental phenotype, which can only be verified after death and dissection. Thus critical matings to test hypotheses become almost impossible. In spite of these difficulties, Promptoff (1929) proposed that three pairs of genes were responsible for variations observed in dorsosacral vertebrae number. The dominant member of one pair produced the lengthened type when either homozygous or heterozygous. But the dominant allele at the second locus was epistatic, resulting in the normal type. The dominant allele at a third locus, which was epistatic to the other two, caused the bantam type.

Kyphoscoliosis. Kyphoscoliosis has been studied over a long period of time at the University of California, first at the Berkeley campus, and then at Davis. Taylor (1971) selected for and against deformed spinal column for over 20 years. The incidence in his high line was over 50 percent and that in the low line was less than five percent. Both curvature of the spine (scoliosis) and an increase in the vertical arch of the synsacrum (kyphosis) were characteristic of his stock. About 90 percent of the scoliotic birds were also kyphotic with the incidence of both traits being greater in males.

In the F_1 generation from crosses of high and low incidence lines no individuals with scoliosis were produced although some kyphosis was still expressed. In F_2 and later generations, the incidence of scoliosis ranged from 2.5 - 23.5 percent. No major genes were identified but Taylor (1971) felt that both scoliosis and kyphosis had some genes in common. Further study at Davis suggested that the etiology of scoliosis was an abnormality in collagen metabolism (Riggins et al., 1977; Lin et al., 1980), and additional genetic studies led to the conclusion that three major recessive autosomal genes were involved. Variation in expression was attributed to incomplete penetrance of the major genes as well as effects from modifying genes and the environment. The observed greater severity among males was attributed to a sex influence on the trait rather than to sex-linkage (McCarrey et al., 1981). A similar condition reported by Czaja (1939) resulting in curvature of the spine and humped back may well have been the same trait. Czaja (1939) attributed its inheritance to several recessive genes since he obtained only 8.75 percent incidence of crippled birds in his crosses.

Spondylolisthesis. Spondylolisthesis, commonly known as kinky back, is a syndrome that only affects broiler chickens (Wise, 1970). The clinical features and pathological anatomy of this condition have been described by Wise (1970). The condition appears between one and four weeks of age. It is characterized by distortion and displacement of the sixth and seventh thoracic vertebrae, with subsequent compression of the spinal cord, resulting in clinical lameness. Chickens with clinical kinky back tend to remain in a sitting position with legs extended forward and all the weight resting on the hocks and tail. When disturbed, kinky back birds struggle backwards on their hocks with their wings flapping. Radiographic studies of the thoracic vertebral column (Wise, 1973) showed that kinky back is not congenital, but it does develop rapidly during the first eight days when growth is rapid. Although the incidence of kinky back is usually low in a population, a much larger proportion of the population may be subclinically affected.

A genetic basis for this syndrome was first suggested by Osbaldiston and Wise (1967), and later Wise (1973) considered that the incidence depends on age, genotype, and rate of early growth. Riddell (1973, 1976) tried to increase the incidence of kinky back in a flock by selecting for the syndrome. By mating affected with affected for three generations, the incidence was increased from 3.8 percent in the first generation to 13 and nine percent in the second and third generations. Likewise, Khan et al. (1977) were able to maintain an incidence of the trait of 58-66 percent in one flock and less than 40 percent in another by selection over four generations.

Dominant rumplessness (*Rp*). Among the various skeletal mutations of the fowl, one of the more striking is the rumpless condition. In the past, this condition has been a characteristic of such breeds as the Persian fowl, rumpless Game Bantams, rumpless Polish, and others (Dunn, 1925). At the present time, it is a breed characteristic of the Araucana. Dunn and Landauer have studied the morphology and inheritance of this trait extensively. Their studies indicated that there are three types of rumplessness: complete, intermediate, and accidental.

In the normal chicken there are 16 synsacral vertebrae which are fused with the pelvis, five free caudal vertebrae, and six vertebrae which are fused to form the pygostyle. The normal chicken also has 14 or 16 tail feathers and a uropygial oil gland. Landauer and Dunn (1925) and Landauer (1928) determined the morphological differences among the three types of rumplessness and the normal state. The complete and accidental forms appeared outwardly to be the same. The posterior of their bodies was round, and they lacked an oil gland and tail feathers. Examination of the vertebrae, however, showed that they were different. The complete rumplessness condition was characterized by a lack of all but the last two pygostyle vertebrae, all the free caudal vertebrae, and one or two vertebrae from the centre of the five synsacrocaudaul vertebrae. The accidental form differed in that all of the pygostyle was absent, and the last two synsacrocaudal vertebrae were missing instead of one or two from the centre. Du Toit (1913) studied the musculature of these birds in detail, and reported that nearly all muscles were present although some were displaced. Landauer and Dunn (1925) felt that some muscles were actually missing. Du Toit (1913) also examined the embryonic development of the complete rumplessness condition and found that the tail vertebrae were absent from the beginning. The intermediate rumplessness differed from both of the others in that all the free caudal vertebrae were present although they were fused together irregularly. As with complete rumplessness, the pygostyle was lacking except for the last two vertebrae, and one or two vertebrae were missing from the centre of the synsacrocaudal vertebrae. Rudimentary oil glands appeared on about 25 percent of this type, and varying numbers of tail feathers (2-17) were usually present.

Davenport (1906, 1909) was one of the first to report genetic studies with rumpless fowl, concluding that a dominant gene was involved. Dunn (1925) verified the dominant status of the complete type, and determined that the accidental type was not genetic. In Dunn's stocks, accidental rumplessness appeared at the rate of about one per 1000 chicks. Dunn and Landauer (1936) assigned the gene symbol *Rp* to the complete type. The genetic basis of the intermediate or modified rumplessness was studied by Dunn and Landauer (1934, 1936) after it appeared following the mating of rumpless to normal. Additional outcrosses as well as selection for the intermediate type resulted in all heterozygotes and some homozygotes of the intermediate type. This phenotype appeared to be due to multiple recessive modifying genes which are carried by normal birds and which tend to suppress abnormal development.

The rumpless condition also had an influence on viability and fertility (Dunn and Landauer, 1934). The proportion of rumpless embryos dying between the 17th to 21st days of incubation was always higher than expected, and the proportion of rumpless chicks that hatched was lower than expected. This applied to both the complete and intermediate types. For the complete type, this reduction in viability was about five percent. They were unable to determine if viability was better in the intermediate type. Increased mortality continued posthatch with 12.1 percent of rumpless and intermediate chicks dying during the first two months as opposed to 7.2 percent of the normal chicks. Fertility problems resulted from purely mechanical reasons. Lack of tails which act as balancers in the copulation act resulted in incomplete copulations. Also very heavy fluff about the cloaca was an additional handicap. Fertility was as low as 17 percent for rumpless x rumpless, and as high as 71.2 percent for normal female x intermediate male. Fertility was always better when the females were normal.

Rumplessness can also be experimentally induced. Landauer (1945b) was able to induce rumplessness in 42 percent of Leghorn embryos by injecting insulin into the egg yolk prior to incubation. Danforth (1932) was able to induce rumplessness in about 7.5 percent of normal chicks by abruptly changing the incubator temperature during the first week of incubation. Likewise, Landauer and Baumann (1943) induced rumplessness in about seven percent of their embryos by mechanically shaking the eggs prior to incubation.

Recessive rumplessness (*rp-2*). A skeletal mutant, found in White Leghorn stock, with many of the features of dominant rumplessness was reported by Landauer (1945a). This condition is commonly known as 'roachback' among poultrymen. The features similar to those of dominant rumplessness were the presence of a rudimentary pygostyle, and free caudal vertebrae which were fused together into a bony knob; in some cases these elements were entirely absent. Depending on the degree of abnormality of the caudal skeleton, the uropygial gland and tail feathers were either absent or the gland was rudimentary with a reduced number of drooping tail feathers. Intermediate conditions ranged from complete rumplessness to entirely normal-tailed. This condition differed from dominant rumplessness in that the fused caudal vertebrae were laterally compressed and bent downward, and the anterior and posterior margins of the synsacrocaudal vertebrae appeared to flare. Synsacral kyphoscoliosis (curvature of the spine and humpback) was also a common feature, and to a lesser degree so were supernumerary ribs. Males showed a definite trend toward more extreme abnormalities than did females. This trait was embryonically determined as early as the fourth and fifth days of incubation (Zwilling, 1945), with the degree of tail reduction being correlated with the time at which abnormal development was initiated. Hatchability was slightly reduced in completely rumpless embryos, and during the posthatch growth period chicks with abnormal caudal skeletons experienced a somewhat increased mortality.

This trait was completely recessive and autosomal, but with low penetrance and wide degree of expressivity. Modifying genes, some of which might have been sex-linked, tended to influence toward normality. When recessive rumpless Leghorns were crossed with breeds which differed in their predisposition to sporadic rumplessness (spontaneous nongenetic), the degree of penetrance and expressivity of the recessive rumplessness condition changed and diverged in the direction of the differences in incidence of sporadic rumplessness of the original stocks (Landauer, 1955). Landauer (1945a) assigned the gene symbol *rp-2* to the main autosomal recessive gene involved with this trait.

VARIANTS OF THE APPENDICULAR SKELETON

Variations in the appendicular skeleton consist of those that affect the legs, toes, and wings. They are listed in Table 7.2. Mutations of the wings are much fewer in number than those affecting the legs.

Polydactyly and duplicate polydactyly (*Po*, *Po*^{d}). Chickens normally have four toes, but some breeds have five (Figure 7.2). It is characteristic of Dorkings, Houdans, Silkies, and Sultans. The fifth toe develops on top of the normal first toe, arising from the metatarsal. It is longer than the first toe. This type of polydactyly is the ideal type as far as the breed standards are concerned, but in actual fact there is considerable variation in the expression of polydactyly. Warren (1944) suggested that there are three distinct developmental steps involved in the expression of this trait. Sometimes these steps are not perfectly executed, resulting in a wide range of expression. The first of Warren's suggested steps was the addition of a long digit above the normal first digit; this was followed by the extra digit splitting; and finally, there was loss of the normal first digit. He was able to rationalize four of the observed phenotypes with this hypothesis. A fifth phenotype which Warren (1944) called 'polyphalangy' consisted of the first toe having an extra phalanx. Although these birds still had only four toes, the polyphalangy condition only appeared in polydactylous stock, and thus appeared to be a modified polydactyly. Polydactyly was present at hatch, and actually appeared as early as the fifth day of incubation (Kaufmann-Wolf, 1908). Unilateral polydactyly, also known as heterodactyly, has been observed by many workers (Bond, 1920, 1926; Punnett and Pease, 1929; Warren, 1944). The number of digits on each foot was different, with the left foot usually having the extra digits and the right foot having the normal four. Also both feet could be polydactylous, but phenotypically different. Polydactyly can also be suppressed by subjecting embryos to low temperatures at 1-4 days of incubation (Sturkie, 1943; Warren, 1944).

Many workers (Bond, 1926; Punnett and Pease, 1929; Landauer, 1948; Warren, 1944) have developed extensive data on the inheritance of the polydactyly trait. Although it is a complex trait influenced by modifier and suppressor genes, the basic mode of inheritance is that of an incompletely dominant autosomal gene. The gene symbol *Po* was assigned to this locus by Dunn and Jull (1927). Warren (1944) further studied the polyphalangy condition in pure polydactylous stock, and concluded that it behaved as if an autosomal

Table 7.2. Listing of inherited variations in the appendicular skeleton of chickens.

Trait and gene symbol		Trait and gene symbol	
Polydactyly	Po, Po^d, po^+	Congenital leg anomaly	cla, Cla^+
Recessive polydactyly	$po\text{-}2, Po\text{-}2^+$	Congenital crippling	coc, Coc^+
Multiple trait semilethal	psp, Psp^+	Congenital perosis	cop, Cop^+
Short-legged Cornish	Cl, cl^+	Ungual osteodystrophy	
Creeper	Cp, cp^+	Tibial dyschondroplasia	
Ametapodia	Mp, mp^+	Micromelia-Hays	$mm\text{-}H, Mm\text{-}H^+$
Shankless	shl, Shl^+	Congenital crooked toes	crt, Crt^+
Abnormal tibial metatarsal joints		Brachydactyly	By, by^+
Crippling anomaly		Sex-linked wingless	wl, Wl^+
Genetic perosis	pe, Pe^+	Autosomal wingless-3	$wg\text{-}3, Wg\text{-}3^+$

dominant controlled it with some unknown factors causing variability in dominance. Landauer (1948) was able, by selection over seven years, to raise the proportion of sinistral heterodactylous birds from 13 to 33 percent, but at the expense of the polyphalangous birds which almost disappeared from his stock. Another complication in the inheritance of this trait was the suggestion that some polydactylous breeds as well as some nonpolydactylous breeds carry suppressor genes that can completely inhibit the expression of polydactyly in genotypically polydactylous birds (Punnett and Pease, 1929; Hutchinson, 1931; Warren, 1944).

Warren (1941) reported on a new type of polydactyly, which breeding tests showed was autosomal dominant and an allele at the *Po* locus. This allelism was further substantiated by Warren (1944) using linkage tests with both the duplex and multiple spur loci. Hutt (1949) assigned the gene symbol Po^d to this allele. This type of polydactyly, which Warren (1941) called duplicate, caused a much greater duplicating of the first digit with a wider variation of expression. The most frequent expressions were a six-toed condition in which a normal first toe and a split extra toe were present (Figure 7.2), and a five-toed condition in which the normal first toe was replaced by a long toe showing varying degrees of splitting. In some extreme cases, extra toes occurred along the shank up as high as the hock joint, while in others an almost complete duplication of the foot and shank were present. Extreme cases usually did not survive. Asymmetry of expression between the two feet was frequently observed. Doubling of the first digit of the wing was evident in duplicate birds but not in ordinary polydactyl birds.

Recessive polydactyly (*po-2*). A recessive type of polydactyly was discovered in Rhode Island Reds in which 22 percent of the affected birds had five toes, 31 percent had six toes, and the remaining 47 percent had the hind toe missing but replaced by two or three extra toes on the shank (Ibe and McGibbon, 1980). Mortality of affected chicks was about double that of the normal sibs. About 25 percent died as late embryos while an additional 17 percent died by six weeks of age. Those living beyond six weeks of age usually showed some leg deformities.

The gene symbol po^r was suggested for this trait by Ibe and McGibbon (1980). However, they did not suggest that this was an allele at the dominant polydactyly locus (*Po*). Thus, subsequently the gene symbol *po*-2 has been assigned to this locus (Somes, 1981).

Multiple trait semilethal (*psp*). McGibbon and Shakelford (1972) reported a rather unusual trait, with a reappearance of it being reported by Smyth (1981). Birds with this multiple-feature trait all showed polydactyly, syndactyly, and ptilopody as a unit. Each of these traits, however, is known as a separate genetic trait, with polydactyly being discussed in this chapter, and syndactyly and ptilopody in Chapter 6. The polydactyly expression in the multiple trait semilethal was similar to that described elsewhere in this chapter, whereas the syndactyly was much more extreme than that usually seen. Webbing was present between each pair of digits and the hind toes were curled inward and forward. The ptilopody was extreme and quite unlike any of the normal shank feathering conditions. In this instance, down feathers covered the whole shank including the bottoms of the feet and the webs between the toes. This trait was also semilethal in that only 25-30 percent of affected chicks hatched, and of those that did hatch most died within three to six weeks. Only one affected bird lived to maturity, a male which was later used in breeding tests.

McGibbon and Shackelford's (1972) mutant appeared from a Buttercup x White Leghorn bantam cross, while Smyth's (1981) mutant came out of a cross between two color

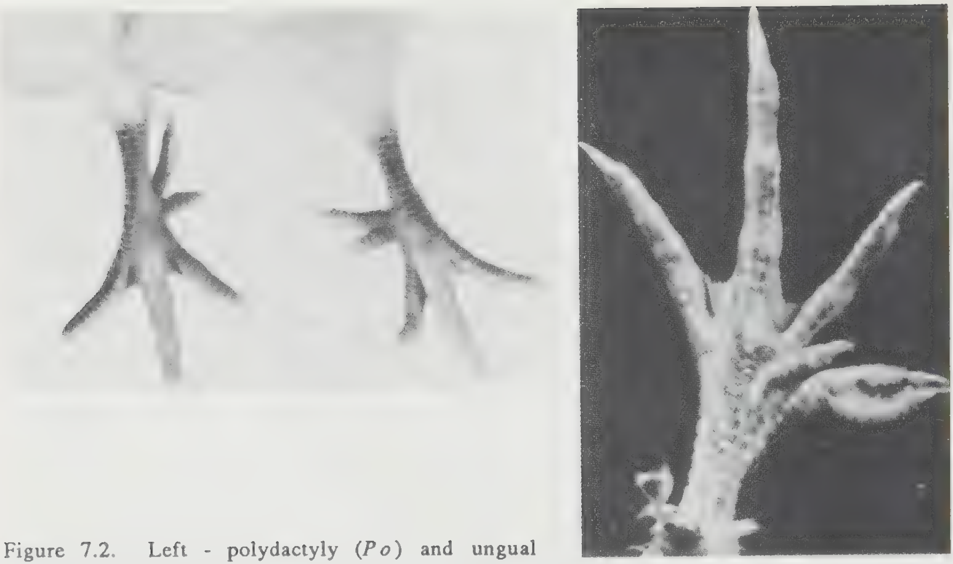

Figure 7.2. Left - polydactyly (*Po*) and ungual osteodystrophy of digit III (from Warren et al., J. Hered. 35:354, 1944). Right - six-toed phenotype common in duplicate polydactyly (Po^d) (from Warren, J. Hered. 32:2, 1941).

stock lines. In neither case did the parents or similar stocks show any of these three traits. Breeding tests of both mutant stocks gave the same results. The semilethal triple-trait complex was inherited as a unit, and was the result of a single autosomal recessive gene, to which McGibbon and Shackelford (1972) applied the gene symbol *psp*.

Short-legged Cornish (*Cl*). The Cornish breed is characterized by a very broad full body with comparatively short thick legs set wide apart. The head is also very broad and short. Landauer (1935a), in studying these conformational differences between Dark Cornish and other breeds, found a lethal factor associated with those birds having the shortest legs. In these short-legged Cornish the tibiotarsus and tarsometatarsus were markedly shortened, and the fibula was overly developed such that it extended to the lower end of the tibia where it fused with the tibial epiphysis. Genetic studies showed that the factor was lethal at the end of incubation when homozygous, but contributed to shortened extremities when heterozygous. Bone measurements (Landauer, 1939a) showed increased proportional reduction of long bones of the limb with increased distance from the body; even the toes were reduced in length.

Crosses between short-legged Cornish and Leghorns produced an F_1 with long bones of intermediate length. In the F_2 generation, leg length ranged from the most extreme short Cornish type to legs longer than those of the Leghorn grandparent. These data seem to indicate that the Cornish leg type is the product of multiple factors, one or more of which are dominant over the appearance of long legs. One of the dominant factors which reduces long bone length is lethal when homozygous. However, all Cornish may not have this gene. It may only be associated with the extremely short-legged Cornish. Of all the possible genes involved in this trait, the one with the lethal action was assigned the gene symbol *Cl* by Hutt (1949).

Creeper (*Cp*). Of all the mutations of the appendicular skeleton, this short-legged condition has received more attention than any of the others. The creeper trait has occurred in many parts of the world. It is the distinguishing characteristic of a number of breeds such as the Jitokko and Chabo of Japan, the Courtes Pattes of France, the Scots Dumpy of the United Kingdom, and the Creepers and Japanese Bantams of the United States and elsewhere. This trait was studied extensively by Landauer and others at the Storrs Agricultural Experiment Station. Refer to the bibliography of descriptive and experimental investigations on gene expression and phenotypic differentiation of heterozygous and homozygous embryos by Landauer (1973) for a detailed review.

Cutler (1925) was the first to observe that adult creepers were always heterozygous. This observation was confirmed by the work of Landauer and Dunn (1930) who found that creeper fowl were heterozygous for an autosomal dominant gene, and that the homozygous condition was lethal with embryos dying early on the fourth day of incubation (Dunn and Landauer, 1926). Thus, true breeding creepers do not exist. Hutt (1933) gave the gene symbol *Cp* to this incompletely dominant gene. Landauer (1942) showed that the short-leggedness of the Japanese Bantam was also caused by this same gene, and Shibuya et al. (1972) have shown the same for the short-legged Jitokko breed.

The main feature of the heterozygous creeper is the shortness of its extremities. The creeper condition belongs to the class of skeletal abnormalities known as achondroplasia or chondrodystrophy. Long bones of the limbs are shortened. Landauer (1934a) found that long bones of males were 18-31 percent shorter and females 13.4-24.4 percent shorter than in their normal sibs. The tarsometatarsus was reduced proportionately more than the other long bones. The heterozygous creeper can be identified embryonically as early as six days of incubation (Landauer, 1932). Detailed studies of osteogenesis in embryos, growing chicks, and adults indicated that endochondral ossification was deficient, while periosteal ossification was normal (Landauer, 1931). Thus, the long bones are shorter and thicker than those of noncreepers. The *Cp* gene also appeared to be lethal to about five percent of the heterozygotes (Landauer and Dunn, 1930). This was evident in backcross matings. An additional increase in embryonic mortality of about five percent was evident from eggs laid by creeper females when compared with their normal sisters. This was attributed to variability in egg shell weight (Landauer and Bliss, 1943). Heterozygous creepers were also shown to be more susceptible to vitamin D deficiency (Landauer, 1934b) and to the toxic effects of selenium (Landauer, 1940) when compared with noncreepers.

Most homozygous embryos die after three days of incubation. At this age they are very retarded in growth, especially in the head and limb buds. This early cessation of growth may be due to the *Cp* gene preventing formation of the vascular system (Cairns, 1941). However, a few homozygotes escape this early lethal stage, but they die just before hatching time. Landauer and Dunn (1930) found that 1.6-2 percent of their creepers developed to this late stage. These late creeper embryos were phokomelic, showing extreme and extensive abnormalities of the eyes, head, and extremities (Landauer, 1933, 1939b). In some creeper matings, some embryos had peculiar abnormalities such as lack of various parts of the legs or wings, misplaced bones in the limbs, and missing vertebrae (Landauer, 1935b). In the Japanese Bantam, up to 93 percent of the homozygous creepers developed to the late stage, and in crosses between creeper fowl and Japanese Bantams, this effect was carried over to the hybrids (Landauer, 1942). Fujio and Shibuya (1974) showed a similar effect using the Jitokko breed, but found different expressions of the lethal action of the *Cp* gene on different genetic backgrounds. They demonstrated that a maternal genetic effect caused the enhanced viability, and suggested that autosomal dominant genes were involved. This viability enhancing factor also depended on length of egg production to be effective, with about 90 percent late embryos appearing during the first week of lay, about 40 percent during the second, and none after that. It would appear that a hen just beginning to lay introduced some substance into the cytoplasm of the egg that lowered the early lethal action of this gene (Fujio, 1976). Landauer (1944) found that the proportion of late embryos could be tripled in creeper fowl stock by incubating the eggs during the first 24 hours at 35.5°C instead of the usual 37.2-37.7°C. Even at this early age, the *Cp*/*Cp* genotype was functional, and by retarding development the embryos were allowed to proceed further.

Ametapodia (*Mp*). Ametapodia, a condition studied by Cole (1967), was in many ways similar to the creeper mutant, but ametapodial birds had much more extremely reduced legs (Figure 7.3). This mutation affected basically the legs and to a lesser extent the wings; otherwise the birds were normal. The tarsometatarsal and carpometacarpal bones were absent, but all toes were nearly normal. With age, various foot bones and digits tended to fuse resulting in little or no articulation. Frequently, the nails would fuse together as a sheet of cornified material. Claws which would reach a length of 10-12 mm on digits II and III of the wings. Ametapodial chicks could be raised to maturity and used for breeding, but their maintenance required extreme care because they had problems with locomotion, being only able to hobble and not being able to maintain a toehold.

Cole's (1967) genetic studies showed that ametapodia was inherited as an autosomal dominant, and that all ametapodial individuals were heterozygous. As with the creeper gene, homozygotes were embryonic lethals, in this case the peak of lethality being at the seventh day of incubation. Heterozygotes showed no signs of increased embryonic mortality or reduced hatchability. The gene symbol *Mp* was assigned to the condition. A linkage test with the rose comb locus showing crossing over of 16 percent, while the creeper locus is very closely linked (0.4 crossover units) to rose comb. Thus, although both creeper and ametapodia are on the same chromosome, they would appear to be at separate loci.

Shankless (*shl*). A most unusual mutation associated with a pericentric inversion in chromosome 2 was reported by Langhorst and Fechheimer (1985). This unique limb abnormality was always associated with the homokaryotype for the inversion. The mutant condition, known as shankless, was characterized by malformed and reduced metacarpals, absence of tarsometatarsal shanks, increased tibia length, extra bones in digits two, three, and four, and fusion of proximal phalanges. Birds heterokaryotypic for the inversion were

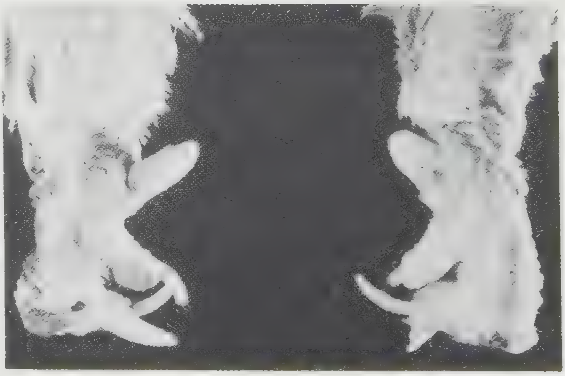

Figure 7.3. Ametapodia (*Mp*). Feet of a yearling male. (From Cole, J. Hered. 58:144, copyright 1967 by Am. Genet. Assoc.).

normal except for a slight reduction in metacarpal length. This condition was generally lethal during late incubation or shortly after hatching. However, some shankless birds did survive to maturity and were used in breeding tests. Fertility of shankless males was similar to that of most other males.

This mutation generally expressed itself as an autosomal recessive, the only exception being the intermediate shortening of metacarpals in the heterozygotes. The gene symbol *shl* was assigned to it. Because of the cotransmittance of the inversion and the mutant, it was postulated that the mutant locus was either located within the inverted chromosome segment or at a breakpoint. One particular advantage in studying this mutant was the ability to identify the heterozygotes through their karyotypes.

Abnormal tibial metatarsal joints. Serfontein and Payne (1934) were among the first to suggest that some types of leg disorders might have an inherited basis. The condition they studied appeared between three and eight weeks of age. First symptoms were squatting, with puffiness and enlargement of the saphenous vein. As the condition progressed, the tibia and tarsometatarus began to bend and became curved, with the gastrocnemius tendon often slipping from its normal position. Leg muscles would sometimes lose their ability to flex and became fixed in an extended position. Heavy breeds seemed to be more susceptible than lighter breeds.

When their normal-legged Rhode Island Reds were mated together, 18.6 percent of the progeny had crooked legs, while crooked-legged matings produced 50 percent of the progeny with crooked legs. Although the mode of inheritance was not evident from their study, it did show that the tendency toward having crooked legs was inherited and probably was polygenic.

Crippling anomaly. Morejohn (1955) reported a heritable crippling anomaly that affected the pelvis, joints of the legs, and the eyelids which were wrinkled in severely affected birds. This mutant was found in a Dark Cornish strain, and all matings were made between presumed carriers of the trait because affected birds did not live beyond 90 days of age. Restricted flexion of the tibial metatarsal joints affected both legs equally. Affected legs underwent a gradual outward twisting and rotation, and the toes curled inward and outward. In only one case did the Achilles tendon slip out of its groove. In some cases, birds were able to regain use of their legs, but their toes always remained curled. Distortions were noted in the pelvic structure, due probably to compensatory muscular forces attempting to maintain the normal stance of the bird.

Crosses between the original Dark Cornish male and female, their offspring, and the Dark Cornish male mated to a New Hampshire female, all suggested that this condition was the result of a single autosomal recessive gene. However, the F_2 generation from the Cornish x New Hampshire cross only produced three affected birds out of 70. Thus, Morejohn (1955) concluded that this trait was inherited, but that its basis was more complex than that of a simple single gene.

Genetic perosis (*pe*). Somes (1969) studied a perosis condition in a randombred meat control population which was maintained on a complete diet throughout the study. These birds developed the condition between three and five weeks of age. Expression of the perotic condition varied widely. Usual expression started with enlargement of the hock joint, followed by varying degrees of twisting of the tibiotarsus and bending of the tarsometatarsus. In extreme cases, the leg would become fixed in an extended position. One or both legs were affected, and quite frequently one or both legs would improve by eight weeks of age to become normal. This type of perosis was shown to be inherited as a single autosomal recessive with a homozygous penetrance of about 74 percent. It was assigned the gene symbol *pe* and shown to be located on chromosome 1.

Congenital leg anomaly (*cla*). This leg anomaly found in a Plymouth Rock strain was evident at hatching, and was a permanent crippling condition in which the head of the femur did not articulate into the acetabulum (Morgan, 1963). One or both legs were involved with crippled legs extending laterally, forward, or upwards. An unusually large proportion of these cripples were males (83 percent) and none survived to maturity. In the unselected flock, only seven percent of the progeny were crippled, while 18 percent were crippled when the parents were known carriers. When outcrosses were made, the trait failed to be expressed. Morgan (1963) concluded that a single recessive gene involving modifiers was the cause of this condition. Somes (1980) applied the gene symbol *cla* to this particular type of crippling.

Congenital crippling (*coc*). Somes (1970) reported a congenital crippling condition which appeared in a White Plymouth Rock x New Hampshire series of crosses. This condition appeared with several other abnormal traits but seemed to segregate separately from them (see blistered foot lethal in Chapter 6). At hatching, one or both legs projected laterally making it extremely difficult for the chick to move around. Livability was very poor for these birds because they had difficulty getting to food and water. In many ways this condition resembled a similar condition reported by Morgan (1963). However, in this instance the birds were not sexed, and thus it was not known if there was a preponderance of males as in Morgan's case.

The genetic studies seemed to indicate that an autosomal recessive gene was involved with possible modifiers. An early mating of carriers produced 38 percent affected chicks, while two later matings of carriers produced only ten and 14 percent affected chicks. In four series of carrier x noncarrier matings, only four out of 239 progeny were affected. The gene symbol *coc* has been assigned to this trait (Somes, 1980).

Congenital perosis (*cop*). Bohren (1951), as cited by Jull (1952), studied a congenital perosis condition in which the newly hatched chicks from certain females mated to a carrier male had twisted legs. Affected chicks died within a few days after hatching. This leg condition was reported to have been due to an autosomal recessive gene for which Somes (1980) assigned the gene symbol *cop*. This trait would seem to be similar to those reported by Somes (1970) and Morgan (1963).

Ungual osteodystrophy. Ungual osteodystrophy, a mutation involving only the toes, was studied at both Cornell University and Kansas State College, and reported in a joint publication (Warren et al., 1944). Expression of the condition was quite variable, but generally it was expressed as a loss of the toenails and terminal phalanges with the third digit being most frequently affected (Figure 7.2). Toes on one or both feet were affected, and in some cases, nails were misplaced and attached to the side of the toe. Genetic studies at both institutions showed that this trait had a complicated multiple factor inheritance. In the F_1 generation, eight percent of the progeny were affected. Affected x affected produced only 47.5 percent affected, and affected x normal F_1 produced 14.6 percent affected progeny.

Tibial dyschondroplasia. This condition, which is known as focal osteodystrophy in the United States and as tibial dyschondroplasia elsewhere, is a leg abnormality caused by cartilage hypertrophy of the tibial and metatarsal bones of broilers. It has been reported from several countries. The condition is characterized by a cone of cartilage produced by the proximal growth plate of the tibia. This cartilage hypertrophy results in enlargement of the hock joint, lameness, and various leg abnormalities (Siller, 1970).

Leach and Nesheim (1965, 1972), by means of family selection, were able to develop high (41 percent) and low (16 percent) incidence lines, with the high line being easily altered through dietary manipulation. Riddell (1976), using individual selection and radiography, also developed high (51 percent) and low (0 percent) lines with the high line being influenced by diet, thus confirming earlier work (Leach and Nesheim, 1965, 1972). Sheridan et al. (1978), using individual selection, developed a high line from which they suggested that a major recessive sex-linked gene was involved. There also appeared to be a high maternal component or dominant genetic component or both involved. Thus, a multifactoral inherited basis seemed to be responsible for this condition with dietary modifiers being rather prominent.

Micromelia-Hays (*mm-H*). Hays (1944) reported a lethal mutation in Rhode Island Reds which segregated as 54 abnormal chicks out of a total of 172. The characteristics of this trait were quite uniform. The head was wide and flat, and both mandibles were greatly reduced but normal in shape. Long bones of both the legs and wings were greatly shortened and thickened, but all leg bones were straight. Hatched chicks struggled around on their abdomens because of the shortness of their legs. About 30 percent of these abnormal chicks hatched, but all died within one week. Normal sibs also experienced greater than normal mortality, suggesting that the gene may also be lethal to some heterozygotes. The inheritance of this trait was assumed to be recessive because of the closeness of fit of the above data to a ratio of 169.5 normal to 56.5 abnormal. The gene symbol *mm-H* has been assigned to this locus (Some, 1980).

Congenital crooked toes (*crt*). A morphological defect of the toes known as crooked or curly toes is not uncommon. Such environmental factors as riboflavin deficiency, or brooding management factors such as smooth floors or crowding under the hovers, have been shown to cause this trait. It has also been shown that there is a genetically determined crooked toe trait that is indistinguishable from that caused by environmental conditions.

Hicks and Lerner (1949) selected for and against crooked toes over a period of five years. Their low incidence line was usually less than two percent affected for the whole period, while their high incidence line reached 97 percent affected at the end of the five years. Fertility and hatchability were not reduced in the high line, even among birds with extreme expression of the trait. These workers did not suggest a mode of inheritance for this trait. Fischer (1956), also working with this trait, concluded that in his Kedu chickens the crooked toe condition was transmitted as a single autosomal recessive, equally inherited in both sexes, but with incomplete penetrance. Somes (1980) assigned the gene symbol *crt* to this locus. Fischer (1956) also studied a similar condition in Australorps, but in that breed he indicated the mode of inheritance was very low. Thus, it would appear that modifying factors which influence the penetrance of crooked toes vary from one breed to another, but that the basic genetic factor for crooked toes is an autosomal recessive gene.

Brachydactyly (*By*). Brachydactyly in the chicken is a condition which affects the outer toe. Normally the fourth toe (the outer one) is 10-12 percent longer than the second toe, but in brachydactylous individuals the fourth is equal to or shorter than the second. The fourth toe normally has five phalanges and a toe nail, but in brachydactylous individuals some of these phalanges may be shortened or completely absent, with the most extremely brachydactylous toes consisting of only two phalanges and no toe nail. Warren (1940) indicated that males tend to show a slightly greater degree of the more extreme variations of these traits. Brachydactyly is commonly associated with feathered shanks (Danforth, 1919; Jaap, 1939; Warren, 1940) and is a common feature of such breeds as Brahmas and Cochins. Warren (1940) found an inverse relationship between the relative length of the fourth toe and the degree of shank feathering, those with the greatest shank feathering having the shortest fourth toe.

Because of the close relationship between brachydactyly and shank feathering, Schmalhausen (1934) proposed that the same genes were responsible for both traits, and that growth of feathers on the shanks interfered with normal development of the fourth toe. Goetinck tried to test Schmalhausen's hypothesis with epithelial-mesenchymal interaction studies (Goetinck, 1967) and breeding experiments (Goetinck, 1971). The first studies showed that these two traits could develop independently, and the latter, though less conclusive, along with the first argued heavily against an associated developmental basis. Warren (1940) proposed from the results of a series of breeding experiments that brachydactyly was inherited as an incompletely dominant autosomal gene, and Hutt (1949) later assigned the gene symbol *By* to this trait. Warren's work showed that those birds with the nail and some phalanges missing were always homozygous for the trait. Those with complete but shortened fourth toes were usually heterozygotes, but a few homozygotes also showed this somewhat reduced expression. The heavy type of shank feathering seems to be due to two genetic loci (see Chapter 6). Warren (1940) suggested that possibly one of these shank feathering genes and the brachydactyly gene were the same. Danforth (1929) felt that both shank feathering genes were able to produce brachydactyly. Serebrovsky (1926) stated that his 'tip type' of shank feathering was always associated with brachydactyly of the outer toe. He also suggested that a dominant gene that he called 'susu' may give rise to the tip type shank feathering. In the tip type, the shank was feathered, but the middle toe was almost bare except for a few feathers on its outer side. This would appear to agree with Warren's (1940) suggestion that one of the shank feathering genes and the brachydactyly gene are the same.

Sex-linked wingless (*wl*). Chickens without wings are indeed unique, but just such a type of chicken was reported by Pease (1962) and further studied by Lancaster (1968). The first wingless chicks were females. They appeared normal except for being wingless, but in subsequent matings both winglessness and varying degrees of leglessness appeared among the progeny (Pease, 1962). In extreme cases both wings and legs were completely absent. The model phenotype, however, was normal legs but no wings (35.6 percent). A nearly nil penetrance expression (less than one percent) consisted of drooping wings with abnormal flight feathers and occasional supernumerary phalanges (Lancaster, 1968). Thus, expression of this condition was extremely varied, but the wings were always affected. Internal organs were normal, but the breast musculature was underdeveloped, and wingless birds had watery eyes. Hatchability of wingless chicks was reduced by 17 percent, but this varied with the degree of expressivity. The model type was easy to raise and was used in matings. Hinchliffe and Ede (1973) studied the embryonic development of this wingless condition, and reported that the condition arises as a result of the precocious appearance of cell death in the anterior necrotic zone of the limb bud at stage 19, with a progressive extension beyond its normal area during stages 20-23. If hindlimb buds were affected, it was due to a similar but less pronounced effect.

Pease (1962) was the first to suggest a sex-linked mode of inheritance for this trait. Further work by Lancaster (1968) verified this mode of inheritance, and he was further able to link wingless to both the *S* and *K* loci. Lancaster (1968) gave the gene symbol *ws* to this locus, but because *ws* had been previously assigned to the trait wing-spot, Somes (1980) assigned the symbol *wl* to wingless.

Autosomal wingless-3 (*wg-3*). In a one sentence statement, Landauer (1973) mentioned a wingless mutant, with skeletal features similar to the sex-linked wingless, which was viable and inherited as an autosomal recessive. No other details are available. Somes (1980) assigned the gene symbol *wg-3* to this particular winglessness trait.

BODY SIZE VARIANTS

Body size in the domestic fowl varies greatly, from that of the Jersey Giant cock at 6.0 kg to that of the Dutch bantam cock at 0.6 kg. Most of the differences that are seen between various standard breeds or between various bantam breeds are due to multiple factors. However, some major single genes have been found which have major effects on skeletal size. These will be described in this section.

Body size at maturity depends not only on skeletal size, but also on muscle mass, skin, feathers, and fat store. Skeletal size does not vary after maturity, but the other factors can change considerably depending on environmental conditions. Thus, when considering body size and its inherited basis, differences in skeletal dimensions are the prime concern. For studies to be particularly accurate, skeletal measurements should be made rather than using body weight. However, in many studies body weight has been the measurement used because it is much easier to get than bone measurements. Another problem with body size studies is the natural sex dimorphism that exists in this trait, with males being larger than females. Some studies have used conversion factors to equalize male and female weights (Punnett, 1923; Waters, 1931).

One of the major features that distinguishes one breed from another is body conformation. Although many breeds have similar body conformation, there are very distinct differences between various breeds and very distinct body conformations associated with each breed. Leghorns certainly differ from Cornish, which differ from Rhode Island Reds, which differ from Modern Games for example. This is a hard trait to quantitate and it can be quite deceiving because loose vs. close feathering can make similar body conformations look quite different (Kopec, 1926). Body conformation is influenced not only by skeletal dimensions, but also by muscle mass. What little information there is on the inheritance of body conformation is presented at the end of this section.

Inheritance of breed size differences. In crosses between breeds in which the size differences were not extreme (a difference of twice as much or less), such as Golden-Pencilled Hamburg x Silver Sebright (Punnett and Bailey, 1914), Silver Spangled Hamburg x White Cornish (May, 1925), White Leghorn x Light Brahma (Waters, 1931), Silky x White Leghorn (Lauth, 1935), and Faverolle x Buff Leghorn (Lauth, 1935), the outcomes were generally the same with some minor differences. The size of the F_1 progeny was between that of the parents or slightly nearer the larger parental breed. In the F_2 generation variability was greater than that seen in the F_1, and in large F_2 populations the number of progeny that were obtained of the parental sizes gave an indication as to the number of genes involved. These results would be expected if this trait was dependent on multiple factors. About 12 percent of the F_2 male progeny obtained by Punnett and Bailey (1914) were equal to or exceeded the parental weights. This high a percentage of extreme individuals in an F_2 population of only 112 birds indicated that not many genes were involved. They proposed that not more than four pairs would give these results. Waters (1931) proposed two pairs of genes from his data on the Leghorn x Brahma cross. Danforth (1929) showed variation between bantam breeds when he crossed a Mille Fleur Belgian d'Uccle bantam male with Sebright females. Some F_1 progeny were larger than the parents, and in later generations birds both larger and smaller than the parental types were obtained. Thus, these results suggested that these two breeds were not similar in size factors although both were bantams.

Dominant sex-linked dwarfism (*Z*). Maw (1935) made reciprocal crosses, which were carried into the F_2 generation, between Light Brahmas and Golden Sebright bantams. The Brahmas were about five times larger than the Sebrights. Their F_1 and F_2 data produced definite evidence that a dominant sex-linked gene was involved in suppressing body weight in the Sebright bantam. The F_1 weights were intermediate between the two parents, but below the midpoint toward the smaller parent. Because the Sebright carried the gold color and rapid feathering genes and the Brahma the silver and slow feathering genes, both located on the sex chromosome, Maw (1935) was further able to classify the birds by using the color and feathering genes to indicate the sex chromosomes of parental origin. Five bones were measured for each of the F_2 progeny. The expected size difference by sex, color, and feather type for the involvement of a sex-linked dominant gene was found.

Jull and Quinn (1931b) also made crosses between breeds differing widely in size (Barred Plymouth Rock and Black Rose Comb bantam with a five-fold difference in size), and had obtained results somewhat similar to those of Maw (1935), but without the added advantage of using other sex-linked genes that were segregating to follow chromosome segregation. Their F_1 progeny were intermediate in size, but like Maw's (1935) they were closer to that of the smaller parent.

Somes (1978) assigned the gene symbol Z to dominant sex-linked dwarfism.

Recessive sex-linked dwarfism (*rg*). Godfrey (1953) made crosses similar to those of Jull and Quinn (1931b) using the Barred Plymouth Rock and Black Rose Comb bantam, but on a much larger scale involving 2399 birds. Like the previous study using these breeds, Godfrey (1953) found the F_1 progeny to have both body weights and shank measurements intermediate to the parents but showing dominance toward the smaller size. Five marker genes were present in the heterozygous F_1 generation, and by backcrossing to New Hampshire females a relationship was shown to be present with both the silver-gold and slow-rapid feathering sex-linked loci, but no relationship was evident with the other three marker genes. Males showed no difference in weight or shank length, while the gold-rapid feathering females were significantly smaller than the silver-slow feathering females. Thus a sex-linked recessive gene or genes located near the color and feathering loci was responsible for part of the small size of the Black Rose Comb bantam. The effect of this gene consisted of a ten percent reduction in size, exhibiting most of its effect prior to nine weeks of age. Godfrey (1953) assigned the gene symbol *rg* to this locus and suggested this might be the same locus that Maw (1935) described as being dominant. With Maw's small F_2 numbers, such an error might be made. Godfrey (1953) further estimated, based on F_1 and F_2 generation variances and parental mean differences, that about 16 pairs of genes were involved in the genetic control of body size differences between these two breeds, with the recessive sex-linked *rg* gene being sufficiently stronger than any of the others to be detected as a single entity.

Sex-linked dwarfism (*dw*, dw^M, dw^B). Hutt (1959) reported a peculiar dwarf condition that appeared in a New Hampshire flock. This condition became the well known and much studied sex-linked dwarfism of the fowl (Figure 7.4). Hutt (1959) determined that this was a recessive sex-linked trait, closely linked to both the gold-silver and slow-rapid feathering loci, and he assigned the gene symbol *dw* to this locus. A tremendous literature now exists relative to this locus (refer to Chapter 20).

This particular gene has a much greater dwarfing effect than any previously discovered. Males are reduced in size by about 43 percent while females are reduced by 26-32 percent. This gene has a truly major effect on size. Its retardation of growth could be observed by as early as two weeks of age, and this slowed rate of growth continued into adulthood. Dwarfs were quite healthy and viable, and their fertility and hatchability was as good as normal birds. Egg size, however, was smaller but only by about ten percent, and egg numbers were slightly reduced. The *dw* gene is not known to be part of the genotype of any bantam breeds, but it would seem to be a good gene to use in the production of a bantam breed.

Two additional alleles have been proposed for the Dw^+ locus. Custodio and Jaap (1973) studied a size reducing sex-linked recessive gene from the Golden Sebright bantam breed which was closely associated with the sex-linked feathering locus. Their gene, which they called 'bantam gene' and to which they assigned the gene symbol dw^B, reduced female size by 5-11 percent when compared to $Dw^+/$-females. Dw^+/dw^B males were reduced by about five percent when compared to Dw^+/Dw^+ males, and dw^B/dw^B males were reduced in

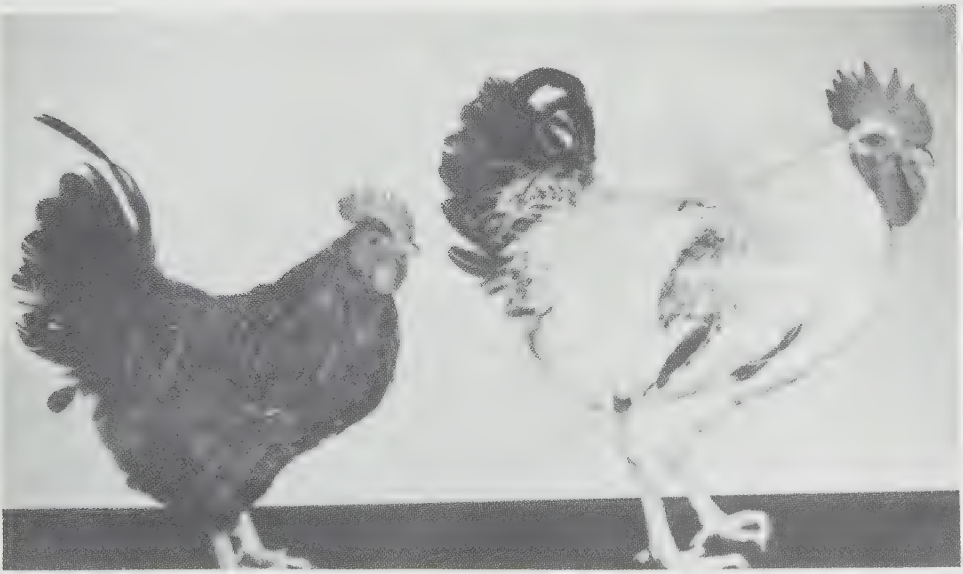

Figure 7.4. Sex-linked dwarfism. Full brothers at 32 weeks of age. Dwarf (dw/dw) on the left, normal (Dw^+/dw) on the right. (From Hutt, J. Hered. 50:213, 1959).

size by about nine percent when compared with Dw^+/dw^B males. Thus, this additive effect of the dw^B gene would imply that males homozygous for this allele would be reduced in size by about 14 percent when compared to normal males. The dw^B allele would seem to be recessive to its incompletely dominant normal Dw^+ allele. These authors also showed that dw^B was dominant over the *dw* allele.

The fourth allele at this locus was proposed by Hsu et al. (1975). The source of their gene was a dwarf isolate from a meat-type line. A set of reciprocal matings showed that the small size was due to a single sex-linked recessive gene. To determine if it was at the Dw^+ locus, they made a cross to a *dw*/-female. The resulting F_1 males were all dwarfed indicating that the two genes belonged to the same locus. Further matings showed that the *dw* and dw^M alleles were in fact different alleles and not the same gene. They designated this allele as Macdonald dwarf and assigned the gene symbol dw^M to it. Tests between dw^M and a dw^B allele obtained from an Old English Game bantam were inconclusive as to whether these two alleles were the same or different. However, the authors concluded that this was probably a new and different allele because dw^B only reduced female body weight by ten percent and shank length by five percent with the birds generally appearing normal, whereas the dw^M allele reduced female body weight by 13.5 percent and shank length by nine percent, and these birds could definitely be distinguished from normal birds by their smaller size. The dominance relationship between this new recessive allele and the other two recessive alleles is unknown.

Autosomal dwarfism (*adw*). A single autosomal gene which affected body size was reported by Cole (1973) to have occurred in the Cornell K strain White Leghorns. Birds were reduced in body size by about 30 percent, and were easily distinguishable by 6-8 weeks of age. These dwarfs had excellent viability and reached an adult weight of about 1400 g. Their sexual maturity was somewhat delayed. Egg production was about 87 percent of normal, with egg size at one year of age being 57 g. Hatchability for these dwarfs was also somewhat reduced. Cole (1973) assigned the gene symbol *adw* to this autosomal recessive dwarf locus. It is not known whether this particular gene is part of the genotype of any of the bantam breeds.

Body conformation differences. One of the first to work in this area was Kopec (1926, 1927) who made reciprocal crosses between White Leghorns and Buff Orpingtons. He concluded that there are genes that affect overall body size, but that there are also genes that independently determine the size of various parts of the body. Maw and Maw (1938) substantiated this by showing that special genes determined the length of the sternum. Cock (1969) showed this to be true for the leg bones from the results of his selection experiments in which he selected four lines for long and short shanks and for high and low body weights. He identified six factors: a factor which increased the leg bones relative to the rest of the body, a factor which increased the length of the tarsometatarsus, a factor which increased the thickness of all long bones, one which affected the degree of flatness or roundness of the shaft of the tarsometatarsus, and another which did the same for the coracoid and scapula, and finally a factor affecting the length of the nasal region and lower jaw relative to the cranium. Possibly some of these factors mentioned by Cock (1969) are the cause of the relatively long legs of the Modern Game breed or the relatively short thick legs of the Cornish breed. In any case, it appears that body conformation is a multifactoral trait resulting from the combined action of body size genes, independent body part genes, and muscle mass genes.

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Chapter 8. GENETICS OF BIOCHEMICAL VARIANTS IN CHICKENS

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The inheritance of biochemical variants is based on the concept of one gene : one enzyme or protein as proposed by Beadle and Tatum (1941). They and others have contributed to knowledge in this area, mainly through investigations of microorganisms. As we understand the concept today, a sequence of nucleic acids on a chromosome codes for a protein which may have enzymatic properties like an esterase, or be nonenzymatic with binding properties such as albumin. A mutation, often a substitution of one nucleotide base for another at a particular site on the chromosome, may result in a substituted amino acid in a protein which may then be detected as a variant. There may be one mutant of the wild-type allele or there may be several such mutant alleles which produce many variant forms of the protein, often referred to as polymorphisms or genetic polymorphisms.

Most biochemical variants in animals were discovered as the result of zone electrophoresis perfected by Smithies (1955). He prepared a gel from hydrolyzed potato starch mixed with a buffer solution, inserted a protein source such as blood serum, applied an electrical potential difference across the gel, and after proteins had migrated for some time, stained the gel with a protein-specific dye. Proteins at a given pH have either a net negative or positive charge, or a net zero charge. The charged proteins will migrate to the anode or cathode ends of a gel and become revealed on the gel slab after staining as zones or bands. Genetically controlled biochemical variants are evident as differences in zone patterns among individual animals. Smithies and Hiller (1959) found genetic polymorphisms for human transferrins and haptoglobins (Smithies and Walker, 1956) in this way. These electrophoretic variants and others are controlled by codominant alleles located on autosomal chromosomes.

The next major advance in zone electrophoresis came with the development of a method to detect enzyme heterogeneity. Hunter and Markert (1957) were the first to use zone electrophoresis with histochemical staining techniques, which had been used for a long time previously for tissue analyses, to show multiple forms of esterase and later lactate dehydrogenase (Markert and Moller, 1959) in animals. These multiple molecular forms of enzymes, called isozymes, exist within single cells of an individual. A detailed treatment of this subject was given by Markert (1968). Using this technique, investigators found genetic variation in isozymes among individuals of a population. Like polymorphisms for proteins, all enzyme polymorphisms discovered in chicken populations are under autosomal allelic control.

Revealing genetic polymorphisms by zone electrophoresis can require modification of the electrophoretic system. Such factors as concentration and pH of gel buffer and bridge buffer (which connects the electrodes to the gel), concentration and type of gel ingredients (starch, acrylamide or agarose), voltage and current applied, temperature and type of stain used, can be modified to achieve optimum resolution of variants. Resolution of genetic variants is based principally on charge and size as well as shape of protein molecules under investigation. Therefore many variants probably go undetected because those differing in amino acid content can have identical charge, size, and shape.

Most of this chapter will describe polymorphisms of electrophoretic variants, found in the easily accessible blood and eggs of the chicken. A few variants such as those of low density lipoprotein were discovered in other ways. Some reference will be made to linkage relationships and associations with production traits, but these subjects will be discussed in detail in other chapters.

Tables 8.1 to 8.3 list the known biochemical genetic variants of chickens described in over 25 years of literature (courtesy R.G. Somes, Jr.). The tables contain the name and source of the protein or enzyme, breeds which are polymorphic, alleles identified, and references. Occasionally the tables list breeds with unique alleles or breeds which each contain only one of the alleles studied. The allelic symbols are mostly those of Somes (1988) which are not necessarily the same as those of the investigators who discovered the variants. When more than one reference is recorded per protein or enzyme, the first one indicates the discoverer of the polymorphism and the subsequent references indicate those who discovered new alleles.

BLOOD, YOLK AND MUSCLE PROTEINS

Before the advent of zone electrophoresis, serum proteins were separated electrophoretically by moving boundary electrophoresis based on the Tiselius system (Smithies, 1955; White et al., 1968). Using an alkaline pH, components of serum or plasma included (from anode to cathode) albumin, α globulin, β globulin (plus fibrinogen if plasma), and γ globulin. Albumin represented more than half of the total serum proteins. Although these four (five) groups of proteins were thought to be reasonably homogeneous, zone electrophoresis with starch, polyacrylamide or agar resolved the proteins into many components (Smithies, 1955). The resolution attained allowed for the revelation of variants of a particular protein differing by amino acid substitutions or attached moieties under the control of a multiple allelic series.

Table 8.1 summarizes the genetic polymorphisms of chicken blood, yolk, and muscle. Most have been detected by zone electrophoresis. Based on electrophoretograms of Tanabe et al. (1981) and those described in the references noted in Table 8.1, proteins listed in order from prealbumin-3 (at the anode end of the gel) to transferrin would be expected to appear approximately in the order listed if a gel buffer of pH 9.2 were used.

Prealbumin. Prealbumins-3, 2 and 1 are each controlled by two autosomal codominant alleles (Table 8.1). The three phenotypes of prealbumin-1 are shown in Figure 8.1. Prealbumin-3 seems to occur in the yolk while the other two appear in both serum and yolk (Tanabe et al., 1981). However, prealbumin-2 does not appear in plasma of cockerels or non-laying pullets unless injected with estradiol and it seems to be phosvitin according to Tanabe et al. (1981). Phosvitin and lipovitellin are two phosphoproteins found in yolk and are the cleavage products of vitellogenin which occurs in liver and plasma of hens or estrogenized roosters (Deeley et al., 1975). Phosvitin migrates electrophoretically in the prealbumin region and is polymorphic (Table 8.1). Prealbumin-1 appears to be part of the livetin fraction of yolk (Stratil, 1970).

Prealbumin-M of yolk was discovered by two dimensional electrophoresis and is controlled by two autosomal alleles, one of which is dominant to the other (Kuryl and Gasparska, 1983). Homozygotes for the recessive allele *Pa-M*$^-$ do not produce detectable protein.

Albumin. Polymorphism of the largest protein fraction in plasma is controlled by at

Table 8.1. Genetic polymorphisms of proteins found in chicken blood, yolk and muscle.

Name	Source	Population*	Alleles	Reference
Prealbumin-3	Yolk	WL	$Pa\text{-}3^{A,B}$	Tanabe and Ogawa (1980a)
Prealbumin-2	Plasma, yolk, white	WL	$Pa\text{-}2^{A,B}$	Tanabe and Ogawa (1980a)
Phosvitin	Yolk	RIR	$Pv^{A,B}$	Tanabe et al. (1988)
Prealbumin-M	Yolk	WL, WPR	$Pa\text{-}M^{+,-}$	Kuryl and Gasparska (1983)
Prealbumin-1 (livetin)	Plasma, yolk	LS	$Pa\text{-}1^{A,B}$	Stratil (1970)
Albumin	Plasma, yolk, seminal plasma	BL, WL, WC, JF, GL, LS	$Alb^{F,S,C,C1,D}$	McIndoe (1962), Stratil (1968a), Baker et al. (1970), Hashiguchi et al. (1981)
ß-livetin	Yolk	WL	$Lt^{F,S}$	Juneja et al. (1983)
Postalbumin-A	Plasma, yolk, white	WL, WPR, PR	$Pas\text{-}A, pas\text{-}A$	Kuryl and Gasparska (1976)
Pretransferrin	Plasma	WL	$Prt^{+,-}$	Juneja et al. (1982a)
Transferrin (conalbumin)	Plasma, yolk, white, seminal plasma	LS, WC, GS	$Tf^{A,B,BW,C}$	Ogden et al. (1962), Stratil (1968b), Baker et al. (1970)
Vitamin D binding protein	Plasma	RIR, NH	$Gc^{F,S}$	Juneja et al. (1982a)
Haptoglobin	Plasma	WR, C	$Hp^{S,F}$	Shabalina (1977)
Complement factor B	Plasma	WL	$C\text{-}B^{F,S}$	Koch (1986)
Hemoglobin	Erythrocyte	ACRB	$Hb1^{A,B}$	Washburn (1968)
Myosin light chain-1	Breast muscle	WL, NH, RIR	$Lc1^{I,II,III}$	Rushbrook et al. (1982), Rushbrook and Somes (1985)
Low density lipoprotein	Plasma	WL, An, etc.	$Lcb^{1,2,0}$	Pesti et al. (1981)
High density lipoprotein	Plasma	WL, RIR	$Lp\text{-}1^{a,o}$	Ivanyi (1975)

*WL = White Leghorn, WPR = White Plymouth Rock, LS = Light Sussex, An = Ancona, WC = White Cornish, NH = New Hampshire, BL = Brown Leghorn, PR = Plymouth Rock, RIR = Rhode Island Red, WR = White Rock, C = Cornish, ACRB = Athens Canadian Randombred, WPR = White Plymouth Rock, GL = *Gallus lafayettei*, JF = junglefowl, GS = *Gallus sonnerati*.

least five autosomal codominant alleles (Baker et al., 1970; Hashiguchi et al., 1981). Albumin plays a major role in osmotic regulation, and serves in a transport function for fatty acids, trace elements, drugs (Martin et al., 1981), and calcium (Urist et al., 1958).

ß-livetin. Juneja et al. (1983) subjected this water soluble protein from yolk to two-dimensional zone electrophoresis to separate the ß-livetin fractions from albumin, and showed three phenotypes controlled by two codominant alleles (Table 8.1). The fast and slow patterns were each characterized by a row of five major and minor fractions and the heterozygote was a composite of the two patterns.

Postalbumin-A and pretransferrin. Postalbumin-A was shown to be present or absent in all females but always present in males. Inheritance is controlled by two autosomal alleles, one of which is dominant (Kuryl and Gasparska, 1976). Homozygous recessive females do not have the postalbumin. Since all males had the postalbumin zone and none were found without the zone, even from matings where such males were expected, Kuryl and Gasparska (1976) hypothesized that homozygous recessive male embryos died.

Pretransferrin is also under the control of two autosomal alleles with dominance (Table 8.1). Juneja et al. (1982a) used two-dimensional electrophoresis to reveal the polymorphism in which the homozygous recessive genotype resulted in no detectable pretransferrin.

Transferrin. This protein is synthesized in the liver, binds iron, and is identical in amino acid composition to conalbumin which is synthesized in the oviduct and is found in egg white (Lush, 1966). Three phenotypes are shown in Figure 8.1. Ogden et al. (1962) verified that polymorphism of both transferrin and conalbumin were controlled by the same autosomal codominant alleles, even though each allele produces three transferrin fractions but only one conalbumin fraction. Williams (1962) treated transferrin with neuraminidase to remove sialic acid and found that the three fractions were reduced to the slowest for each

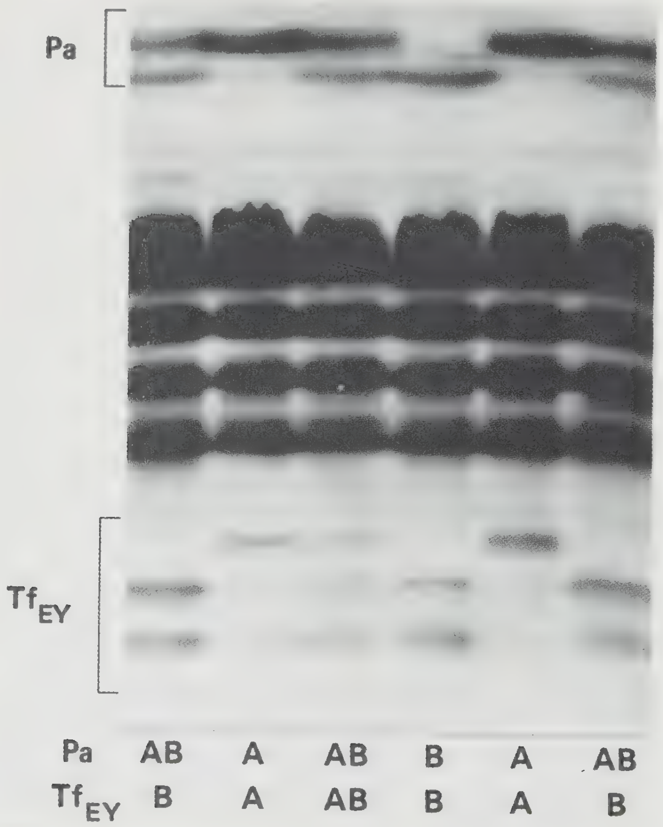


Figure 8.1. Starch gel electrophoretogram of chicken egg yolks showing polymorphic phenotypes of both prealbumin-1 (*Pa*) and transferrin (Tf_{EY}). (From Stratil, 1970).

allele, and that fraction had the same mobility as conalbumin which does not have sialic acid.

Vitamin D binding protein. Genetic polymorphism for this protein was revealed by means of the same two-dimensional zone electrophoresis procedure used to find pre-transferrin (Juneja et al., 1982a). Two autosomal codominant alleles control this polymorphism. The symbol *Gc* has been used to designate alleles of this protein because the same symbol was used for the analogous protein in mammals, i.e. group-specific component. Juneja et al. (1982b) have also shown that the loci for albumin and vitamin D binding protein are closely linked in chickens.

Haptoglobin. This serum protein binds hemoglobin. Shabalina (1977) described a polymorphic system of this protein controlled by two autosomal codominant alleles.

Complement component factor B. Complement refers to a group of factors present in fresh blood serum or plasma that combines with antigen-antibody complexes to promote the lysis of cells. The avian complement system is not as well characterized as the mammalian complement system but does have a factor B which is involved in both Ca^{++}-dependent and Ca^{++}-independent complement pathways (Koch, 1986). When factor B was subjected to agarose gel electrophoresis, Koch (1986) observed a polymorphism under the control of two autosomal codominant alleles (Table 8.1). Homozygotes are each characterized by four zones, while the heterozygote is characterized by six zones. In mammals, the comparable factor B is linked with the major histocompatibility complex, but this was not the case for chickens (Koch, 1986).

Hemoglobin. This protein carries oxygen from lungs to tissues throughout the body. Washburn (1968) used zone electrophoresis on cellulose acetate to show a major and a minor but slightly faster migrating zone. A still faster migrating variant of the minor zone was of quite low frequency in the Athens-Canadian Randombred broiler strain. Except for the related Ottawa Meat Control strain, Washburn (1976) failed to find the polymorphism in several birds and strains surveyed. The polymorphism is under autosomal codominant control. Callegarini et al. (1969) have shown that the polymorphism in the minor hemoglobin fraction is due to an amino acid substitution in the γ-globin chain, as opposed to the α-globin chain which is also part of the protein. The allelic designation of Garcia (1979) has been adopted.

Myosin light chain-1. This protein is in the fast white fibres of the pectoralis major muscle of chickens. Using polyacrylamide gel electrophoresis, Rushbrook et al. (1982) and Rushbrook and Somes (1985) described three electrophoretic zones controlled by three autosomal codominant alleles (Table 8.1). The locus for this polymorphism is not linked to the muscular dystrophy locus.

Low density lipoproteins. These lipoproteins are part of the lipid transport system and have a density between 1.006 and 1.063 g/ml. Pesti et al. (1981) developed alloimmune precipitating sera against low density lipoproteins to find the polymorphism. Three autosomal alleles Lcb^1, Lcb^2 and Lcb^o were hypothesized, whereby Lcb^o is recessive to the other two alleles and probably produces a low density lipoprotein for which there is no reagent.

High density lipoproteins. These lipoproteins have a density between 1.063 and 1.21 g/ml. Ivanyi (1975) produced allotypic antisera against high density lipoproteins in response to injections of *Bordetella pertussis* into chickens. Birds were typed as positive or negative for the high density lipoprotein antigen which can be considered to be under the control of a dominant allele (Table 8.1).

EGG WHITE PROTEINS

Although three of the blood and yolk proteins listed in Table 8.1 are also present in egg white, those listed in Table 8.2 seem to be exclusive to egg white. They are presumed to be synthesized by the magnum. The five egg white proteins are listed in Table 8.2 in the order in which they might appear in a polyacrylamide gel with anode at the top (Figure 8.2) and with a buffer of pH 9.0 (Tanabe and Ogawa, 1980b). Lysozyme is not evident in Figure 8.2 because it does not show well using many standard gel buffers, most of which are above pH 7.0 (Baker et al., 1970). The names of the ovoglobulins, G_1 to G_4, are those given by Longsworth et al. (1940).

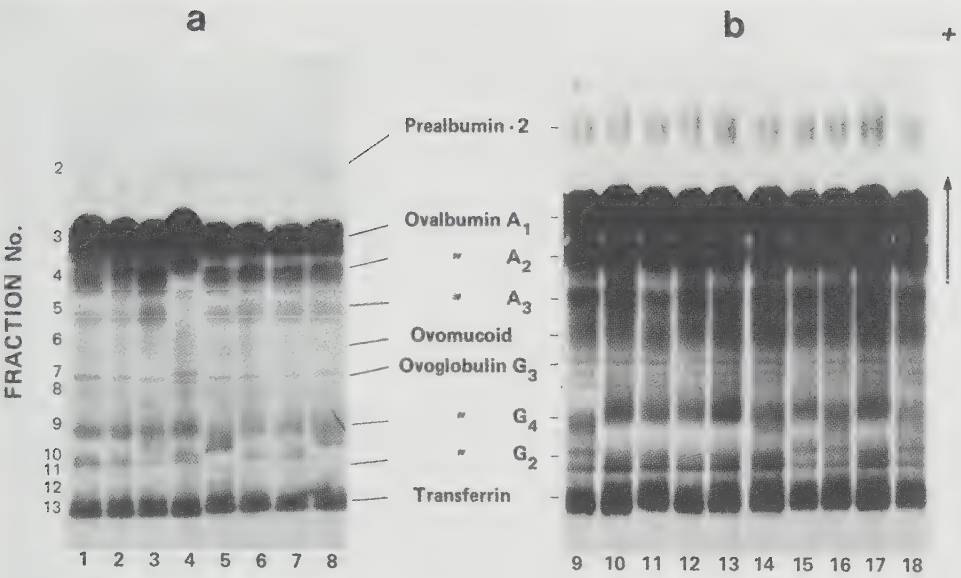
Ovalbumin. Lush (1961) was the first to describe genetic polymorphisms in egg white proteins. Using starch gel electrophoresis and a gel buffer of pH 8.65 he found two variants of ovalbumin called B for faster and A for the slower more commonly found variant. Genetic control is by two autosomal codominant alleles. Croizier (1966) found a new ovalbumin type called F in the Faverolle breed. The three zones associated with the product of each allele are the result of two, one or zero phosphate residues per molecule (Baker and Manwell, 1962; Lush, 1964). The fastest migrating zone has two phosphate residues per molecule and therefore it is the most negatively charged. However, even after removal of phosphatases, unexplained heterogeneity remains. Observed quantitative variation may have a genetic basis not associated with the *Ov* locus (Baker and Manwell, 1962).

Ovoglobulin G_3. This protein, originally designated II by Lush (1961) migrates slightly more slowly than ovalbumin in a weakly alkaline starch gel (Baker et al., 1970).

Table 8.2. Genetic polymorphisms of proteins found in chicken egg white (albumen) and characterized by zone electrophoresis.

Name	Source	Population*	Alleles	Reference
Ovalbumin	White	BL, F, etc.	$Ov^{A,B,F}$	Lush (1961), Croizier (1966)
Ovoglobulin G_3	White	BL, RIR, JF, etc.	$G_3^{A,B,J,M}$	Lush (1961), Baker et al. (1970)
Ovoglobulin G_4	White	WL	$G_4^{A,B}$	Tanabe and Ogawa (1980b)
Ovoglobulin G_2	White	BL, WL, RIR, GL	$G_2^{A,B,L}$	Lush (1961), Baker et al. (1970)
Lysozyme (ovoglobulin G_1)	White	WL, PB	$G_1^{F,S}$	Baker (1968)

*BL = Brown Leghorn, RIR = Rhode Island Red, JF = junglefowl, WL = White Leghorn, PB = Polish Bantam, GL = *Gallus lafayettei*, F = Faverolle.



	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Prealbumin 2	–	B	B	AB	A	AB	–	–	–	–	–	–	–	–	–	–	–	–
Ovalbumin	AB	AB	A	B	A	A	A	A	A	A	A	A	A	A	A	A	A	A
Ovoglobulin G_3	AB	AB	B	A	B	B	B	B	B	B	B	B	B	B	B	B	B	B
Ovoglobulin G_4	A	A	A	A	B	A	A	AB	B	A	A	A	A	AB	A	AB	A	AB
Ovoglobulin G_2	B	B	AB	B	A	B	B	A	AB	B	B	B	B	B	AB	AB	B	AB
Transferrin (Conalbumin)	B	B	B	B	B	B	B	B	B	B	B	B	B	B	B	B	B	B

Figure 8.2. Polyacrylamide gel electrophoretograms of chicken egg white showing the regions of prealbumin-2, ovalbumin, ovoglobulin G_3, G_4, G_2, and transferrin/conalbumin and phenotypes below where classified. For electrophoretograms a and b, .13 M Tris-borate buffer at pH 9.0 and .98 M Tris-borate buffer at pH 9.0 were used, respectively. (Photo courtesy H. Tanabe).

They have observed up to ten G_3 variants but found that four, A, B, J and M, are easily differentiated using weakly alkaline buffers, while others exist depending on flocks sampled and conditions of gel buffers. Buvanendran (1967) has shown that the ovalbumin and ovoglobulin G_3 loci are tightly linked.

Ovoglobulin G_2. This ovoglobulin has similar appearing zones to those of ovoglobulin G_3 but migrates in general more slowly than zones of G_3 in slightly alkaline gels (Figure 8.2), and the two proteins can be differentiated by heating egg white or by adding iron or silver nitrate to it (Baker et al., 1970). In addition to the two autosomal codominant alleles postulated by Lush (1961), Baker et al. (1970) identified another, G_2^L, which originated from junglefowl (*Gallus lafayettei*).

Ovoglobulin G_4. A third polymorphic ovoglobulin protein called G_4 was discovered more recently by Tanabe and Ogawa (1980b). The variants of G_4 migrate between those of

G_3 and G_2 using horizontal polyacrylamide gel electrophoresis and a gel buffer of pH 9.0. Two autosomal codominant alleles were described (Table 8.2).

Lysozyme. Baker (1968) found a polymorphism in lysozyme or ovoglobulin G_1 which hydrolyzes the polysaccharide outer layer of many airborne gram-positive bacteria (Martin et al., 1981). Most birds have the F phenotype but the S variant has been observed in a flock of Polish bantams (Baker et al., 1970). These authors stated that resolution is best using a gel buffer with pH 8.4 barbiturate, or pH 4.7 to 5.7 acetate whereby the protein migrates cathodally.

ENZYMES

There are 26 enzymes of chickens with genetic polymorphisms which have been demonstrated by zone electrophoresis (Table 8.3). Unlike the polymorphisms of proteins summarized in Tables 8.1 and 8.2, enzyme polymorphisms generally cannot be detected using protein stains because the concentration of enzyme protein is too low and because of other interfering proteins. Enzyme zones are more easily detected using a histochemical stain similar to that of Hunter and Markert (1957). For enzymes such as esterases and phosphatases, the staining solutions will contain a substrate and a diazo dye for attachment to part of the hydrolyzed substrate to reveal enzyme activity as a colored zone or zones on the gel. Cations, cofactors, and other substances, including specific inhibitors for classification purposes, may be in the staining solution. The reader should refer to references in Table 8.3 for specific staining procedures appropriate to each enzyme.

Alkaline phosphatase. Phosphatases hydrolyze esters of phosphoric acid and alchohols using buffers of about pH 9. The first genetic polymorphism of an enzyme in chickens was observed in serum alkaline phosphatase. Law and Munro (1965) and Wilcox (1966) observed a fast or a slow zone in individual electrophoretic patterns under the control of a dominant (*Akp*) and a recessive (*akp*) allele. This polymorphism also exists in liver (Tamaki and Watanabe, 1979). Birds with the fast zone possessed genotype *Akp/Akp* or *Akp/akp* and birds with the slow zone had genotype *akp/akp*. This was the first example of autosomal dominant inheritance of protein or enzyme electrophoretic patterns in chickens. Law (1967) studied zymograms of leucine aminopeptidase of chicken plasma and observed a constant leading zone, and a two-zone phenotype which migrates slightly slower than that of alkaline phosphatase, but with a parallel inheritance. This was confirmed by Csuka and Petrovsky (1972). Treatment of plasmas with neuraminidase, which removes terminal sialic acid from mucoproteins, resulted in the faster variant migrating at the same rate as the slow variant for both enzyme systems, but no change in the migration rate of the slow variant. The constant zone of leucine aminopeptidase was also slowed by the neuraminidase treatment. Law (1967) hypothesized a third gene which possesses a dominant allele for adding sialic acid to each of the protein products of genes for each of the two enzymes to produce the fast variants. Wilcox (1966) observed that the level of alkaline phosphatase activities was greater for 6-week-old birds with fast, compared with slow, alkaline phosphatase variants. This result was observed in 32-day-old chicks for alkaline phosphatase (Tamaki, 1975) and for leucine aminopeptidase (Tamaki, 1976). Tamaki (1975) confirmed the results of Law (1967) and showed that removal of sialic acid did not influence the alkaline phosphatase activity of plasma of birds with either phenotype. More

Table 8.3. Genetic polymorphisms of enzymes found in various chicken tissues and characterized by zone electrophoresis.

Name	Source	Population*	Alleles	Reference
Acid phosphatase	Liver	WL	Acp, acp^{o}	Tanabe and Tamaki (1966)
Acid phosphatase-2	Liver, kidney, spleen, leucocyte	WL, NH, WR	$Acp\text{-}2^{A,B}$	Okada & Hachinohe (1968)
Adenosine deaminase	Erythrocyte	WL, MNF	$Ada^{A,B,C}$	Shotake et al. (1976), Grunder & Hollands (1978)
Alkaline phosphatase/leucine aminopeptidase	Plasma, liver	WL	Akp, akp	Law and Munro (1965), Law (1967)
Alkaline phosphatase-2	Plasma	BPR, RIR, RIW	$Akp\text{-}2^{o}, akp\text{-}2^{a}$	Kimura et al. (1979)
Amylase-1	Plasma	WL, BL, WC, etc.	$Amy\text{-}1^{A,B,C,D}$	Hashiguchi et al. (1970), Tanabe et al. (1977), Hashiguchi et al. (1986)
Amylase-2	Pancreas	WL, NH, OEDG, WL	$Amy\text{-}2^{A,B,C}$	Heller and Kulka (1968), Lehmer & Malacinski (1975)
Amylase-3	Plasma	BDF, JF	$Amy\text{-}3^{A,O}$	Maeda et al. (1987)
Carbonic anhydrase	Erythrocyte	WR, WC, NH, RC, WL	$Ca\text{-}1^{A,B,C}$	Ahlawat et al. (1984)
Catalase	Erythrocyte	L, NH, RIR, PR, F, etc.	$Ct^{A,B}$	Shabalina (1972)
Esterase-1	Plasma	MT, WL, WPR, NH, JF, LNF	$Es\text{-}1^{A1,A2,B,C,D}$	Grunder (1968), Kuryl et al. (1986), Hashiguchi et al. (1986)
Esterase-2	Plasma	WL	$Es\text{-}2^{A,O}$	Kimura (1970)
Esterase-3	Liver	WL, WPR, NH	$Es\text{-}3^{A,B,O}$	Okada and Sasaki (1970)
Esterase-4	Liver	WL, SD	$Es\text{-}4^{A,B,O}$	Tanabe and Ise (1972)
Esterase-5	Liver	WL, WPR, NH	$Es\text{-}5^{A,B}$	Okada (1973)
Esterase-6	Liver, etc.	WL, SD	$Es\text{-}6^{A,B}$	Tanabe and Ise (1972)
Esterase-7	Liver	Mk	$Es\text{-}7^{A,O}$	Asanoma and Tanabe (1974)
Esterase-8	Erythrocyte	WL, In, RIR, Fa, etc.	$Es\text{-}8^{A,B}$	Hashiguchi et al., (1979)
Esterase-9	Liver	WL,NH, Au	$Es\text{-}9^{A,O}$	Ueda and Hachinohe (1973)
Esterase-10	Cardiac muscle	WL	$Es\text{-}10^{A,B}$	Kimura (1975)
Esterase-11	Cardiac muscle	WL	$Es\text{-}11^{A,B}$	Kimura et al. (1978)
Glyoxalase 1	Erythrocyte	WL	$Glo^{1,2}$	Rubinstein et al. (1981)
Mannosephosphate isomerase	Liver, muscle	WL	$Mpi\text{-}1^{A,B,C}$	Kimura et al. (1981)
6-Phosphogluconate dehydrogenase	Erythrocyte	MNF, INF	$Pgd^{A,B,C}$	Shotake et al. (1976), Hashiguchi et al. (1981)
Phosphoglycerate kinase	Erythrocyte, sperm	WL, Au	$Pgk^{F,S}$	Cam and Cooper (1978)
Phosphoglucomutase	Cardiac muscle	WL	$Pgm^{A,B}$	Kimura (1974)

*WL = White Leghorn, NH = New Hampshire, WR = White Rock, BPR = Barred Plymouth Rock, RIR = Rhode Island Red, RIW = Rhode Island White, WC = White Cornish, WPR = White Plymouth Rock, OEDG = Old English Duckwing Game, RC = Red Cornish, L = Leghorn, PR = Plymouth Rock, F = Faverolle, SD = Satsuma Dori, Mk = Japanese strain, In = Ingie, Fa = Fayoumi, Au = Australorp, JF = junglefowl, LNF = Lanka native fowl, INF = Indonesian native fowl, MNF = Malaysian native fowl, BDF = Bangladesh domestic fowl, MT = Meat type, BL = Brown Leghorn.

recently, Washburn et al. (1980) described a third allele, dominant to the other two.

Kimura et al. (1979) reported a second plasma alkaline phosphatase system characterized by the presence or absence of a single zone migrating anodally to the alkaline phosphatase system of Law and Munro (1965). The system called alkaline phosphatase-2 is controlled by two autosomal alleles with dominance; *Akp-2*^{o} is dominant to *akp-2*^{a}, in that the zone is present only when a bird is homozygous for *akp-2*^{a}.

Acid phosphatase. This type of enzyme hydrolyzes phosphate esters at about pH 6.0. The first of two polymorphic acid phosphatase systems of liver was described by Tanabe and Tamaki (1966) and characterized by the presence or absence of a zone according to Okada and Hachinohe (1968). They suggested that the zone was inherited autosomally and that the *Acp* allele was incompletely dominant to the *acp*^{o} allele because the zone of the heterozygote was less intense than that of *Acp/Acp* and no zone was observed for the homozygous recessive genotype.

Okada and Hachinohe (1968) described the second acid phosphatase system as being controlled by two codominant autosomal alleles *Acp-2*^{A} and *Acp-2*^{B}. Because the heterozygote contains an intermediate hybrid zone in addition to the A and B zones observed in homozygotes, each zone is thought to consist of two protein chains known as a dimer and the hybrid zone of the heterozygote would consist of a dimer of the A and B proteins as shown for red cell phosphatase in man (Hopkinson et al., 1964).

Adenosine deaminase. This enzyme catalyses the hydrolysis of adenosine to produce inosine and ammonia. The polymorphic system of erythrocytes is characterized by a fast and a slow zone (Grunder and Hollands, 1978). The zones are controlled by two autosomal codominant alleles (Table 8.3). However, Shotake et al. (1976) published a diagram showing two zones per allele and reported three alleles in Malaysian native fowl populations.

Amylase. α-amylases are found in pancreas and blood. They hydrolyze glycogen and starch and the residual polysaccharides of starch to give glucose, maltose and products which do not give color with iodine. Three polymorphic amylases have been found in the chicken. Amylase-1 (Table 8.3) is under the control of four autosomal codominant alleles (Hashiguchi et al., 1970, 1986; Tanabe et al., 1977). One allele produces one zone. Amylase-2 of the pancreas is also polymorphic and has an inheritance like that of amylase-1 (Heller and Kulka, 1968; Lehrner and Malacinski, 1975). Amylase-3 of plasma is characterized by two zones, controlled by a dominant gene. The two zones are absent in birds of the homozygous recessive genotype (Table 8.3). The recessive alleles have been reported only in Bangladesh domestic fowl and junglefowl (Maeda et al., 1987).

Carbonic anhydrase. This is a zinc-containing enzyme which acts as a decarboxylase for carbonic acid to form carbon dioxide and water. A polymorphism for carbonic anhydrase of erythrocytes was discovered by Ahlawat et al. (1984). The six phenotypes observed were under the control of three autosomal codominant alleles (Table 8.3). No indication was given that the isozymes were inhibited by acetazolamide, a specific inhibitor of carbonic anhydrase (Augustinsson, 1958).

Catalase. Hydrogen peroxide forms water and oxygen under the influence of catalase, an iron-containing enzyme which is found in most animal cells. Shabalina and Russev (1968) noted a genetic variation in quantity of catalase in chicken erythrocytes. Later, Shabalina

(1972) reported electrophoretic variants A and B controlled by two autosomal codominant alleles (Table 8.3). However, the activity and thermostability of the Ct^B/Ct^B genotype was less compared to the Ct^A/Ct^A or Ct^A/Ct^B genotypes.

Esterases. These enzymes catalyze the hydrolysis of ester linkages, although many of the reactions are reversible for synthesis. Simple esterases such as liver esterase catalyze reversibly the breaking and synthesis of ester linkages of lower alcohols and fatty acids. For example, the reversible hydrolysis of ethyl butyrate will produce ethanol and butyric acid.

Esterases have been the subject of a number of investigations by zone electrophoresis. Table 8.3 lists 11 different polymorphic esterases and their controlling alleles which have been discovered in the chicken. Many of these esterases have been classified using various substrates and inhibitors according to methods developed by Augustinsson (1958, 1961) including the use of the inhibitor acetazolamide. This inhibitor is specific for carbonic anhydrase, but does not inhibit esterases, and therefore can be used to differentiate between the two enzymes, even though both may hydrolyze the same ester linkage. The reader may pursue the references of Table 8.3 for more information on classification of each polymorphic esterase. The exact biological substrates are not known precisely. The numbering of esterases in Table 8.3 indicates the chronological order of discovery in general rather than the order of electrophoretic migration.

Esterase-1 of serum was described by Grunder (1968), Csuka and Petrovsky (1968) and Kimura (1969) as having three phenotypes under the control of two codominant alleles at an autosomal locus. Subsequently, three more alleles were discovered (Table 8.3). The

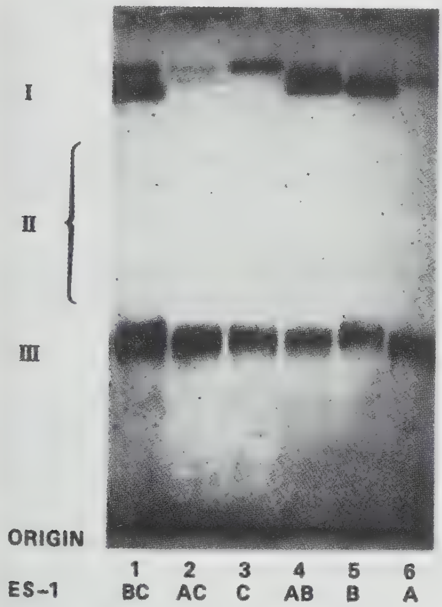


Figure 8.3. Starch gel zymogram of hen sera showing polymorphic phenotypes of esterase-1 (Es-1) in region I. (From Grunder, 1971).

polymorphism of esterase-1 had different phenotypes depending on the zone electrophoresis system used. The Tris (hydroxymethyl) amino methane plus HC1 gel buffer of pH 7.0 used by Grunder (1968) resulted in three zones per allele, while the citric acid gel buffer of pH 8.65 used by Kimura (1969) or a similar buffer used by Grunder (1971) resulted in only one zone per allele (Figure 8.3). There were other differences between the systems but it should be realized that isozymes on the gel may be a result of the *in vitro* conditions and not necessarily reflect biological realities. Polymorphic esterases 5, 6, 8, 10 and 11 are inherited through autosomal codominant alleles as is esterase-1 (Table 8.3). Polymorphic esterases 2, 3, 4, 7 and 9 are inherited differently in that, although controlled by autosomal genes, each system has an allele recessive to the other(s) so that homozygous recessive birds do not produce zones. Esterase-9 is unique because it is one of the very few polymorphic protein systems revealed by urea treatment; the enzymatic activity produced by the dominant allele is resistant to urea. Kimura and Kameyama (1970) have established that esterases-1 and 2 are closely linked.

Glyoxalase. This enzyme, which catalyzes the binding of aldehydes to reduced glutathione, is polymorphic in chicken erythrocytes and under the control of two autosomal codominant alleles Glo^1 and Glo^2 (Rubinstein et al., 1981). The Glo^2/Glo^2 homozygote produces a single zone, whereas the Glo^1/Glo^1 homozygote is almost devoid of activity or produces a faint slower migrating zone. However, the Glo^1/Glo^2 heterozygote produces a dimer which migrates between the faster and the slower zone which is sometimes present.

Mannosephosphate isomerase. This enzyme catalyzes the interconversion of mannose 6-phosphate and fructose 6-phosphate. Kimura et al. (1981) found the polymorphism of this enzyme in liver and muscle to be controlled by three autosomal codominant alleles (Table 8.3).

6-Phosphogluconate dehydrogenase. This dehydrogenase is part of the phosphogluconate oxidative pathway or hexose monophosphate shunt found in animal tissues for metabolizing glucose. The enzyme catalyzes the oxidation of 6-phosphogluconic acid to form D-ribulose 5-phosphate. Shotake et al. (1976) and Hashiguchi et al. (1981) reported a polymorphism controlled by three codominant alleles (Table 8.3). The heterozygote possesses a hybrid zone.

Phosphoglycerate kinase. This kinase is involved in glycolysis for the phosphorylation of ADP. Cam and Cooper (1978) described a polymorphism for this enzyme under the control of two autosomal codominant alleles (Table 8.3).

Phosphoglucomutase. Mutases catalyze the migration of a phosphate group from one hydroxyl group to another of the same molecule. This important glycolytic enzyme was discovered to be polymorphic in cardiac muscle and to be under the control of two autosomal codominant alleles (Kimura, 1974).

CONCLUDING REMARKS

The number of genetic polymorphisms found since the first discoveries in the 1960s has been quite large in chickens as well as in other species of poultry and in mammals. Lush (1966) documented eight biochemical polymorphisms in chickens. Over 20 years later, there are 48 as documented in Tables 8.1 to 8.3. It is recognized that the number of systems may have been overestimated because independent laboratories have not always made comparison tests by exchanging biological materials. It seems certain that many new polymorphisms could be found with additional effort. New discoveries will likely be made, especially with gel electrophoresis because, as Stormont (1970) emphasized, "Gels are fun!"

Past use of polymorphisms. The discovery of so many polymorphisms in chickens and other species has prompted many investigators to seek the reason for their existence. Lewontin and Hubby (1966) addressed this question in relation to population genetics and evolution, but did not arrive at any simple explanation for so much heterozygosity.

The poultry breeder might look on these genetic polymorphisms as an aid to improvement of poultry stocks for economically important traits. The pleiotropic effect of the genes controlling biochemical variants on production traits has been investigated for a number of loci as reviewed in Chapter 20. To the author's knowledge, none of the alleles listed in Tables 8.1 to 8.3 are being used by breeders. Before including biochemical traits in his selection program, the breeder must ask how he will achieve the maximum predicted response for the lowest cost, for instance by selecting the trait directly, by selecting on some index of the trait and a correlated polymorphic trait, or by selecting only for the correlated polymorphic trait.

The identification of new loci always helps to complete the linkage map of the chromosomes. Mention has already been made of certain linkage relationships between loci controlling biochemical polymorphisms. Details of the linkage relationships of all mutant loci are described in Chapter 21.

The biochemical variants have provided new examples of gene action in chickens, although most of the examples can be found in other species also. The polymorphism of alkaline phosphatase, as well as leucine aminopeptidase, is dependent upon the addition of sialic acid by a third locus (Law, 1967). Dimer formation of allelic products has been noted for liver acid phosphatase-2 (Okada and Hachinohe, 1968) and glyoxalase (Rubinstein et al., 1981). Several polymorphic proteins have so-called 'null alleles' which do not produce detectable proteins in gels, for instance prealbumin-M, postalbumin-A, pretransferrin (Table 8.1), alkaline phosphatase-2, esterases-2, 3, 4, 7, and 9 (Table 8.3). Rushbrook et al. (1988) studied the amino acid content of myosin light chain-1. They pointed out that protein variants I and III are shorter than protein variant II, yet the interaction of the N-terminal end of the peptide with actin is not compromised. It would seem that these various forms of gene action are appropriate to the particular function of the gene product.

Future importance of polymorphisms. One of the concerns of the 20th century is the decline in genetic diversity of our animal and poultry populations. It has been pointed out that most of the commercial chicken, turkey, and duck stocks are in the hands of very few primary breeders (see Chapters 2 and 40). One way of addressing the problem is to set aside stocks for potential use in the future as gene resources. One criterion for making the correct choice of stocks to be preserved could be high heterozygosity for biochemical variants.

Perhaps the most important use for genetic polymorphisms is in biotechnology research. Many loci controlling these polymorphisms have or could be placed on linkage maps (Chapter 21) to aid the biotechnologist in locating and transferring specific regions of DNA for these and other loci. The loci controlling these polymorphisms are characterized for number of alleles, and often for differences in their allelic products. Freeman and Messer (1985) have described the challenges of directed gene transfers in domestic fowl. It would seem that genes controlling biochemical polymorphisms are prime candidates for perfecting transfer techniques because the gene product can be identified easily. These loci can be used in studies of gene transfers both intra- and interspecifically to determine effects on production and disease resistance. Biotechnology research should be greatly aided by the knowledge of biochemical variants about which some of the gene action is known.

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Chapter 9. MUTATIONS AND MAJOR VARIANTS OF THE NERVOUS SYSTEM IN CHICKENS

R.D. Crawford

To date, twenty mutations or variants of the nervous system in chickens have been reported. This number is rather large in relation to the number of variants known to affect other body systems of the bird. The nervous system is not necessarily more prone to genetic change. It is likely instead that any genetic change will result in modification of normal behavior of the affected individual, making it noticeable to the investigator and arousing his curiosity. Since behavioral derangement usually has negative effects on growth and egg production, investigation of the defect to remove it from the breeding population is often warranted. Some of the abnormal behaviors have been investigated because of their similarity to disease conditions in man and other animals, and they have become valuable animal genetic models for human diseases and defects of the nervous system.

There is a very large and specialized literature concerning the normal anatomy, neurophysiology, and biochemistry of the avian brain and nervous system. The standard reference resource is Pearson's (1972) *The Avian Brain*. Also of interest to poultry scientists is the collection of reviews in Ookawa's (1983) *The Brain and Behavior of the Fowl*. Publication of Kuenzel and Masson's *A Stereotaxic Atlas of the Brain of the Chick* is keenly awaited; it should stimulate further research into defects of the nervous system.

In this chapter, the genetic aspects of nervous system variants in chickens will be reviewed. They will be described under four headings. There are ten variants that result in tremors of the body; where detailed investigations have been made, nearly all of these have been shown to involve degeneration of Purkinje cells in the cerebellum. The congenital loco or star-gazer syndrome was the first anomaly to be described in the literature (Knowlton, 1929), it is the most widespread in chickens and also in other poultry species, and it is probably the least understood of all nervous system defects. Seven of the variants result in convulsive seizures; two of them, epileptiform seizures and paroxysm, have had prolonged and intensive investigation at least partly because of their potential as animal genetic models of human epilepsies. And one mutant, tipsy, appears to involve neuromuscular function and is not related to the other variants.

MUTATIONS CAUSING BODY TREMORS

The ten mutant conditions resulting in tremors of the body are listed in Table 9.1.

Five of them (cerebellar hypoplasia, cerebellar degeneration, shaker, sex-linked nervous disorder, and jittery) are attributed to sex-linked recessive genes, they all have very similar phenotypic expression, and they all have a defect of the cerebellar Purkinje cells. Only regarding jittery and shaker is there good genetic evidence that separate loci are involved. There is strong likelihood that one or more of the five represent reoccurrence of the same mutation. Description of these five is as follows.

Table 9.1. Mutant conditions resulting in tremors of the body.

Inheritance and trait name	Gene symbol	Reference
Sex-linked recessive		
Cerebellar hypoplasia	*ce*, *Ce*$^+$	Winterfield (1953)
Cerebellar degeneration	*cd*, *Cd*$^+$	Markson et al. (1959)
Shaker	*sh*, *Sh*$^+$	Scott et al. (1950)
Sex-linked nervous disorder	*sln*, *Sln*$^+$	Kawahara (1955)
Jittery	*j*, *J*$^+$	Godfrey et al. (1953)
Autosomal recessive		
Shaker-2	*sh-2*, *Sh-2*$^+$	Somes (1978)
Congenital tremor		Hutt and Child (1934)
Hereditary nervous disorder	*hnd*, *Hnd*$^+$	Okano (1980)
Congenital quiver	*cq*, *Cq*$^+$	Smyth et al. (1985)
Faded shaker	*fs*, *Fs*$^+$	Silversides and Smyth (1986)

Cerebellar hypoplasia (*ce*). A peculiar hypoplasia and degeneration of the cerebellum associated with a nervous disorder was described by Winterfield (1953). It was found in a number of commercial hatchery supply flocks. They had been derived from two breeder flocks of New Hampshire females mated to males from a breeder of R.O.P. New Hampshires. The nervous disorder was evident about 12 weeks of age, and it was seen only in pullets. It consisted of uncontrollable bobbing or anterior-posterior palsy-like movements of the head and cervical region, suggestive of a cerebellar involvement. Incidence varied from 3-10 percent in the 15 flocks inspected. Growth and livability of affected birds were normal, and they reached sexual maturity, but they were discarded before the breeding season.

Upon necropsy, it was found that affected birds had a distinctly rudimentary cerebellum, indicating hypoplasia or atrophy (Winterfield, 1953). The cerebellum was only 12.5-25 percent of normal size for mature birds, and only half the size of the cerebellum in a normal day-old chick. Pathological changes were restricted to the cerebellar cortex. Molecular and granular layers were very thin. Many neurons had undergone degenerative changes, and there was a decrease in numbers of Purkinje cells. All tests for presence of disease were negative.

No breeding tests could be conducted. However all evidence suggested the presence of a sex-linked recessive gene. Somes (1980) assigned the gene symbol *ce* to the condition.

Cerebellar degeneration (*cd*). Markson et al. (1959) reported a defect of the cerebellum in Light Sussex pullets which they believed was comparable if not identical to the cerebellar defect described by Winterfield (1953) in New Hampshire pullets. It occurred only in females and usually appeared between two and three months of age. It was characterized by mild ataxia and tremors of the head and neck. Occasional rippling tremors could be seen in birds at rest. When disturbed, they became moderately ataxic, placing their feet wide apart to gain balance as the tremors became more persistent and more marked. Growth and livability were normal and the birds were able to sustain egg production.

A very detailed description of gross and microscopic anatomy was published by Markson et al. (1959) based on 26 affected specimens. The only gross abnormalities were in the cerebellum. It ranged from about half of normal in size to a shrunken nodule, but distinctive shape and gross structure were always present. Microscopic changes were also found only in the cerebellum and included destruction of Purkinje cells. All findings indicated degeneration and atrophy, and not hypoplasia. Field studies showed that only females were affected by the syndrome. It could be reproduced under laboratory conditions in daughters of carrier sires. Affected females were mated to heterozygous males, producing nine female progeny of which six were affected, and six male progeny none of which was affected. It was speculated that no affected males were produced because the gene may be lethal in double dose. The field and laboratory data combined indicated that the condition was caused by a sex-linked recessive gene. Somes (1980) assigned the gene symbol *cd*.

Shaker (*sh*). The shaker condition described by Scott et al. (1950) was found in Rhode Island Reds. The symptoms were similar to those of Newcastle disease and of infectious avian encephalomyelitis, but both diseases were ruled out as causes of the syndrome. The nervous disorder could not be detected until the birds were about four weeks of age. It began as a mild but rapid movement of the head and neck, which became increasingly severe with advancing age. Few affected birds could walk without stumbling after ten weeks of age. Excitement aggravated the condition. Most affected individuals died from starvation, but a few females were reared to maturity.

Fourteen affected birds were examined histopathologically (Scott et al., 1950). There was a characteristic loss of cerebellar Purkinje cells, with degeneration of many of those remaining. The cells to be destroyed last were those of the gyri nearest the base of the cerebellum. Degeneration was indicated by shrinkage and by increased intensity of staining of the cells. The severity of symptoms varied directly with the extent of Purkinje cell destruction.

A total of 467 chicks was obtained from matings of heterozygous sires with normal dams. None of the 226 sons was affected. Among female progeny, there were 113 normal and 128 shaker individuals. These results clearly indicated that the condition was caused by a sex-linked recessive gene. The gene symbol *sh* was assigned to the condition by Mueller (1952).

Sex-linked nervous disorder (*sln*). Kawahara (1955) described another nervous disorder, in Barred Plymouth Rocks, characterized by a tremor of the head and neck. It was seen in some individuals at day-old but not until four weeks of age in others. All of the original affected birds were females. By twenty weeks of age about half of them were unable to stand and walk, and they eventually died. The others reached sexual maturity but their egg production performance was poor. Histological examination revealed degeneration of the Purkinje cells, occurring first in the gyri region of the cerebellum. Degeneration of Purkinje cells was also characteristic of the shaker mutant described by Scott et al. (1950), but in that condition cells in the gyri region underwent failure much later than those in other areas of the cerebellum.

This condition was found to be sex-linked and recessive. Kawahara (1955) mated heterozygous males with normal females and observed 21.1 percent affected daughters; all of the sons were normal. Affected F_1 females mated to heterozygous males produced 31.1 percent affected males and 24.4 percent affected females. Despite segregation deficiencies, Kawahara (1955) concluded that the condition was due to a sex-linked recessive gene. Somes (1980) assigned the gene symbol *sln* to it. However, Cole (1961) has expressed the view that this condition is identical to shaker (*sh*) except for slight differences in expression that could be explained by modifying genes. Only direct tests for allelism could resolve the matter.

Jittery (*j*). The anomaly called jittery arose independently in White Leghorns and New Hampshires (Godfrey et al., 1953). Affected day-old chicks were characterized by retraction of the head over the back, and by rapid lateral vibration of the head. The intensity of both was variable. If they fell over, they could not right themselves. Excitement increased the intensity of both symptoms. Most of the chicks died of starvation within a few days of hatching, and only a few females survived to sexual maturity. These mature survivors tended to move in circles and could not coordinate movements of the head and neck; excitement made them worse.

Histological examination indicated that jittery chicks had a marked deterioration of Purkinje cells of the cerebellum (Godfrey et al., 1953). These cells appeared to be fewer in number than normal, and some of the existing cells stained more intensely than usual. There was little relationship between severity of physical symptoms and the amount of deterioration of Purkinje cells. These findings are similar to those of Scott et al. (1950) for the shaker condition, so that both mutants may share the same physiological basis.

When first discovered, the jittery mutant from New Hampshires was more severely expressed than that from the White Leghorn source, but with succeeding generations the difference in expression disappeared (Godfrey et al., 1953). By mating tests it was shown that the two mutants were the same. The condition was controlled by a sex-linked recessive gene. It was given the name jittery and the gene symbol *j*. Linkage tests with barring (*B*) and with rate of feathering (*K*) loci showed independent assortment. Mueller (1952) had shown that the very similar shaker mutant was located about 26 map units from *K*, which provides good evidence that shaker and jittery are caused by mutations at different loci, even though their phenotypic effects are alike.

There are five tremor conditions controlled by autosomal genes. Three are very similar to the sex-linked kinds. Two others are quite different. Their descriptions follow.

Shaker-2 (*sh-2*). A short description is given in Somes' (1978) *Registry of Poultry Genetic Stocks*, and in some subsequent editions, of another shaker condition, called shaker-2 with the gene symbol *sh-2*. This condition was found by the late W.H. McGibbon, but a full report was never published. By four weeks of age a mild but rapid movement of the head and neck could be observed in affected birds. With increasing age the shaking became more severe. Excitement worsened the condition. Most of the affected birds were unable to stand by 14 weeks of age and death from starvation usually resulted. Unlike the shaker mutant described by Scott et al. (1950), shaker-2 was autosomal. It appeared to be controlled by a single recessive gene but penetrance was low.

Congenital tremor. Hutt (1932) first reported this condition under the name 'congenital palsy', because some chicks affected with tremor had toes that were curled under and partially paralyzed. Later, Hutt and Child (1934) recognized that many of the less extremely affected birds had no sign of paralysis and so the term 'congenital tremor' was adopted. It was found in White Leghorns. Affected chicks had all degrees of tremor, from only barely perceptible to such extreme trembling that the bird could not stand. The tremor was not induced by sudden shock nor was it increased by excitement. Tremors were continuous when the chick was standing, but disappeared when it sat. Most affected chicks could be identified on the hatching tray or when placed in brooders. Rate of vibration was 10-16.5 per second in chicks 1-2 days old. Only a few birds survived the first week, and trembling in them diminished with advancing age. Growth was severely retarded.

Hutt and Child (1934) reported that 408 normal and 39 affected chicks were obtained from matings known to yield chicks with tremors. This ratio of 10.5:1 indicated that the condition was inherited and that it was recessive. They also had evidence that it was autosomal and not sex-linked. They considered the possibility of control by two pairs of genes, but rejected that theory in favor of a single autosomal recessive gene with unusually low penetrance because of the effects of modifying genes. No gene symbol was assigned. Their data were re-analyzed by Sittmann (1967), using methodology of human genetics, who concluded that penetrance of the condition was complete and that its inheritance depended on two pairs of autosomal recessive genes. Somes (1978, and subsequent editions) lists congenital tremor as multifactoral.

Hereditary nervous disorder. A brief report was published by Okano (1980) describing a nervous disorder found in Gifu Native chickens. Affected chicks were characterized by trembling and lateral head shaking. They had difficulty in eating and drinking, and most of them died within a week of hatching. Similarity to the congenital tremor in chicks reported by Hutt and Child (1934) was noted.

Affected chicks were obtained only from sires and dams which carried the defect (Okano, 1980). Progeny from two sires and four dams, all known to be carriers, segregated into four classes: 21 normal males, 23 normal females, 7 affected males, and 7 affected females, which meets almost exactly the expectation if sires and dams are heterozygous for an autosomal recessive mutation. Somes (1988) has assigned the gene symbol *hnd* to this anomaly.

Congenital quiver (*cq*). The condition called congenital quiver, found in Fayoumis, is quite different from other inherited tremors, although it too involves loss of Purkinje cells in the cerebellum (Smyth et al., 1985). It was apparent at hatching as a rapid vibration of the whole body. From 2-6 weeks of age it was difficult to identify. The extent of tremor in older birds was quite variable, ranging from a slight quiver to pronounced shaking. There were a number of pathological changes in the brain, including extensive degeneration of fibre tracts and neurons across the neuroaxis, diffuse loss of Purkinje cells in the cerebellar cortex, spongy rarefaction of the white matter, and perivascular cuffing. The latter two defects were evident in affected chicks immediately posthatching, indicating an embryonic origin for the disorder with progressive spread in the neuroaxis over time. Congenital quiver was suggested as being a potential animal genetic model for a number of neurodegenerative disorders.

Smyth et al. (1985) found that the condition was caused by a recessive gene with complete penetrance. They mated heterozygous males to normal females to determine whether sex-linkage was involved. If it were, then it would be expected that half of the daughters would be affected. They obtained 22 female progeny, all of them normal, indicating that the gene was autosomal. They assigned the gene symbol *cq*.

A curious pleiotropic effect of the *cq* gene results in sterility of affected males (Smyth et al., 1985). Females and heterozygous males had normal reproductive ability. Histological examination of testes from homozygous males revealed cellular disorganization and a marked reduction in spermatogenic activity. An association of infertility and tremor syndromes is known in mice, and there are two other instances of association between reproduction and nervous system function in chickens; pirouette (*pir*) males are delayed in reaching sexual maturity, and the cerebellar degeneration (*cd*) mutation is associated with reduced fertility and hatchability (see Smyth et al., 1985 for review).

Faded shaker (*fs*). The tremor mutant described by Silversides and Smyth (1986) is quite different from all the others. It causes a tremor associated with dilution of feather color. It was found in Light Brown Leghorns. Mortality of affected individuals was very high, and no specific cause could be identified; most of them died between the 18th day of incubation and one week of age; a few survived to three months of age. The tremor was most pronounced in the posterior end of the chick and was constant over time within an individual; it had a frequency of 6-10 vibrations per second. No defects of the Purkinje cells were identified, but there was a very obvious deficiency of myelin in the medulla of the cerebellum. Similar myelination defects are known in mice, all of them associated with posterior tremors.

The feather color of faded shaker chickens ranged from almost normal to nearly white, but it was constant over several feather generations within an individual (Silversides and Smyth, 1986). Melanosomes of affected individuals were deficient in number and they were incompletely melanized. This is the only mutation in chickens known to involve both nervous system and pigmentation. The faded bronze (*fb*) mutation in turkeys exhibits a similar association of defects (see Chapter 12), as does the dark feather nervous disorder (*dn*) in Japanese quail (see Chapter 13). There are many examples of association between coat color and nervous system function in mice and other mammals (see Silversides and Smyth, 1986 for review).

Faded shaker is inherited as an autosomal recessive gene (*fs*), with incomplete penetrance resulting from action of a single dominant gene masking the effects of the *fs/fs* genotype (Silversides and Smyth, 1986).

CONGENITAL LOCO OR STAR-GAZER

The condition known as congenital loco, or in popular terminology as star-gazer, was the first defect involving the nervous system to be reported in the poultry genetics literature. Knowlton (1929) provided a lengthy and detailed description of it in Barred Plymouth Rocks; his is still the primary reference source. According to him, Durant had noted and illustrated the same or a similar condition a few years earlier in White Leghorns.

According to Knowlton (1929), affected chicks hatched normally, but reanalysis of his data by Sittmann et al. (1965) revealed a 15 percent reduction in hatchability. Abnormal behavior becomes evident immediately. The head is drawn back and to one side with the beak pointing upward (Knowlton, 1929; Hutt, 1949), hence the name star-gazer. The chick falls laterally or backwards and lies on its side or on its back. It has great difficulty in righting itself. If it does, the opisthotonus and tumbling are repeated immediately. The condition is nearly always lethal within a few days of hatching because of food and water starvation.

Congenital loco is very widespread in chickens. Knowlton's (1929) study concerned Barred Plymouth Rocks, and he referred to Durant's observations of the condition in White Leghorns. As part of his study, Knowlton (1929) conducted a survey among workers at North American experimental farms and stations. Congenital loco was reported from 30 locations. It was known in White Leghorns, Rhode Island Reds, Barred Plymouth Rocks, White Plymouth Rocks, White Wyandottes, and Anconas. Hutt (1949) also noted the widespread occurrence of the condition. Indeed most poultry geneticists can cite occurrence of the defect in at least several stocks with which they have worked.

Knowlton (1929) concluded from his research that congenital loco was caused by an autosomal recessive gene. He obtained 461 normal and 146 affected chicks from matings of carriers *inter se*, which closely matched the expected 3:1 ratio. The gene symbol *lo* was assigned by Hutt (1949).

It seems peculiar that a defect so widespread as congenital loco should have had so little detailed study. Except for the recent work of Bernier et al. (1987) on ataxia in Delawares, there seems to have been no further research reported since Knowlton's (1929) study. Each generation of poultry science students has been made aware of the condition and taught to cull affected chicks on the hatching trays without further attention. It might be speculated that more than one new mutation has been discarded on the assumption that all star-gazing chicks represent yet another occurrence of Knowlton's (1929) congenital loco. Indeed, the very first chicks exhibiting epileptiform seizures (*epi*) were callously destroyed at hatching by the author on this assumption; only when a few chicks had convulsions in the brooding pens was it realized that a new anomaly had been revealed.

Ataxia in Delawares. Bernier et al. (1987) have reported briefly on studies of ataxia, in a line of Delawares, which have spanned many years. It is not clear whether their description of 'an ataxic (congenital loco) line' means that they have the classical congenital loco syndrome or a different condition. The line had been selected for ataxia; incidence reached 78 percent when crossed with unrelated genotypes, indicating that it was dominant. Both ataxic and normal sibs had defective otoconia (ear dust) in the inner ears, which was interpreted to indicate that the ataxia had limited penetrance. The otoconia were a fused mass, rather than scattered crystals. The ataxia gene was isolated anew from the base population, using defective otoconia as the selection criterion. Dominance of the trait was confirmed, but it had only five percent penetrance, which was what had been encountered when the first selected line was started.

Congenital loco in other species. Star-gazing that is nearly identical to that in chickens was reported in turkeys by Cole (1957). It too was controlled by an autosomal recessive gene, to which Cole (1957) assigned the same gene symbol, *lo*. Gross and microscopic examination of the brains from loco poults gave no indication of the cause of the defect. It was suggested that a study of the semicircular canals and their ampullae might reveal the anatomical basis for congenital loco.

Congenital loco is also known in Japanese quail, but there is also an inherited condition in that species which is called star-gazing (see Chapter 13). Congenital loco in that species has been given the same gene symbol (*lo*) as used for the condition in chickens and turkeys, and star-gazing has the gene symbol *sg*. According to Savage and Collins (1972), critical tests have not been made to determine whether the two mutants are the same or whether they are alleles at the same locus. They cited personal communication from Erway indicating that no structural abnormality was found on macroscopic examination of inner ear morphology of star-gazing (*sg*) Japanese quail, but there were extensive defects in the semicircular canal of the inner ear in Japanese quail with congenital loco (*lo*).

Further studies of ear anatomy and function should be encouraged in all species affected with congenital loco.

MUTATIONS CAUSING CONVULSIVE SEIZURES

The seven mutant conditions resulting in convulsive seizures are listed in Table 9.2. Five of them have received only limited study, but paroxysm has had intensive investigation, and epileptiform seizures, which has been the subject of more research than any of the others, is now regarded as an excellent animal genetic model of human grand mal epilepsies. Descriptions of these seven variants follow.

Crazy (*cy*). McGibbon (1973) briefly described a nervous disorder in newly hatched Ancona chicks from an inbred line. They exhibited violent uncontrolled body movements and lack of balance. A spasm lasted for several seconds and then the chick would come to rest on its back or sometimes on its side. Several chicks in a group would create almost constant turmoil since they were seldom at complete rest. Affected chicks seemed unable to feed and drink. Even when fed with a syringe none survived more than a few days. Absorption of the yolk sac seemed to be normal. Affected chicks were very jaundiced at time of death. Further description of the seizure pattern caused by this mutant would have been of interest. Unfortunately the mutant did not persist in the Ancona line and it is presumed to have been lost (Bitgood, 1989, personal communication).

Analysis of breeding records indicated that the condition was due to an autosomal recessive gene, and the gene symbol *cy* was assigned (McGibbon, 1973).

Pirouette (*pir*). McGibbon (1974) found a behavioral mutant having almost normal viability in a USDA randombred Red population. Affected chicks whirled or pirouetted in small circles when taken from the hatching baskets, and that behavior persisted. The whirling episodes were brief, lasting only about ten seconds. Causative stimuli were not identified. The chicks were able to eat and drink normally and rearing of them was successful. The whirling behavior continued into adulthood. Some of the affected birds also displayed opisthotonus as they grew older, suddenly pulling or jerking their heads over their backs. This behavior was especially prevalent after drinking water, so that leakage of water from the beak stained the feathers of the back and saddle region. The birds were also characterized by tameness and lack of fear of people, and they were not aggressive toward nonmutant pen mates. Sexual maturity was reached normally in females but it was delayed in males. Fertility was satisfactory using artificial insemination, but natural mating was rare.

More recent studies of pirouette (Bitgood et al., 1987a, 1987b) have confirmed these phenotypic effects, noting that day-old chicks also exhibit varying degrees of tremulous head movements. When combined with naked neck (*Na*), it was found that some pirouette chicks had severe lacerations of the neck, where there was no down covering, presumably from uncontrolled rubbing against the broken egg shell during the hatching process.

Table 9.2. Mutant conditions resulting in convulsive seizures.

Inheritance and trait name	Gene symbol	Reference
Sex-linked recessive		
Sex-linked lethal	xl, Xl^+	Goodwin et al. (1950)
Paroxysm	px, Px^+	Cole (1961)
Autosomal recessive		
Crazy	cy, Cy^+	McGibbon (1973)
Pirouette	pir, Pir^+	McGibbon (1974)
Arched neck		Conner and Shaffner (1953)
Arched neck seizures	ans, Ans^+	Nunoya et al. (1983)
Epileptiform seizures	epi, Epi^+	Crawford (1970)

Breeding records and test matings indicated that the mutant condition was due to an autosomal recessive gene. Its expression was the same in both the original line and in outcrossed birds. McGibbon (1974) suggested use of the gene symbol *pir*.

Sex-linked lethal (*x1*). Goodwin et al. (1950) published a short description of a nervous system defect which was found in White Leghorns. It was never given a name, and has continued to be known only as sex-linked lethal and by the gene symbol *x1*.

When it was first discovered, the condition was seen to affect females only (Goodwin et al., 1950). It was hyperacute and affected birds died within a few hours of the appearance of symptoms. Sick chicks were noticed early in the morning following a long period of darkness. They would be extremely listless, and resting on the keel with the tip of the beak touching the floor. Sometimes a complete coma followed, and sometimes dyspnea and tetanic spasms were seen. A few pullets survived several attacks, and several lived to maturity. They produced sons which were presumed to be homozygotes since they exhibited the same symptoms and mortality as seen in hemizygous affected females. Postmortem examination revealed no significant lesions or cause of death. In a later report (Cole, 1961) it was noted that penetrance of the condition had declined, and expression had become much less severe, so that identification of genetically affected birds was not always possible. The interpretation was that accumulation of modifying genes had masked the action of the causative gene.

Results of mating tests clearly indicated that the condition was caused by a sex-linked recessive gene (Goodwin et al., 1950). The gene symbol *x1* was assigned. The mutation was known to be on the Z chromosome but its position was never determined. Cole (1961) found evidence that it segregated independently of the barring (*B*) locus but further attempts at mapping were abandoned because penetrance and expressivity of the gene had become greatly reduced.

Arched neck. Two genetic disorders which cause the neck to arch forward and under the body have been reported in the literature. They may have been separate occurrences of the same mutation, or they may have been quite different in genetic cause.

The first to be reported was the arched neck character in New Hampshires, described by Conner and Shaffner (1953). Affected birds were normal in appearance and behavior when undisturbed, except for a forward arching of the neck. But when disturbed the head was drawn completely below the body where it shook rapidly from side to side. This posture could be maintained for a long period if the bird did not lose its balance and fall. Although the symptoms were those of terminal Newcastle disease, no internal lesions indicative of Newcastle disease, leucosis, or nutritional deficiency could be demonstrated. There were no gross lesions evident in the central nervous system. The defect was not expressed until the birds were 2-3 months of age, the earliest recorded instance being at 25 days of age, and the latest at seven months. Death resulted from inability to eat and drink. Egg production and hatchability were not affected until physical condition had deteriorated badly, but fertility of males was very poor. Fertilization could be achieved through artificial insemination, and a few chicks were produced from planned matings.

An affected female mated to her normal half-brother produced two normal and seven affected progeny, including both sexes. These nine were mated *inter se* in several combinations, producing four affected males and six normal females. Based on these findings the authors concluded that the condition was autosomal rather than sex-linked, and that it was due to the action of several autosomal genes. However the available data are so limited that it could equally well be postulated that only one recessive gene was the cause.

The affected stock seems not to have been perpetuated, and so the question of its genetic basis must remain unresolved. Somes (1978, and subsequent editions) has listed the trait as being multifactoral.

Arched neck seizures (*ans*). The second disorder causing the neck to arch forward and under the body was reported by Nunoya et al. (1983) in Fayoumi chickens. It was expressed as a seizure which could be induced by unexpected loud noise, sudden movement, bright light, and other audiovisual stimuli. The startled bird would crouch with eyes closed, bending the neck so that the head was positioned between the legs. It might take a backward step and crouch again. The seizure lasted for 15-30 seconds. The bird opened its eyes, slowly regained its footing, and recovered completely. The seizure could recur at irregular intervals. It could occur in chickens of all ages, but was most frequent at about six weeks of age. Growth rate and reproductive function appeared to be normal. The histopathological lesions observed in brain and spinal nerves included degeneration of small arteries, cellular infiltration in the cerebrum, and demyelination and/or loss of nerve fibres with edema and Schwann cell proliferation in the cervical spinal nerves. The vascular changes were thought to be the cause of the seizures. Lesions in the cervical nerves were positively correlated with clinical history, and therefore were considered to be secondarily due to repeated convulsions.

Genetic investigation provided good evidence that the seizures were determined by an autosomal recessive gene. When both parents were prone to seizures, all of the progeny were likewise affected. Mating of affected with normal produced all normal progeny, and when these were backcrossed to affected birds, they produced 32 normal and 34 affected chicks. Although numbers of progeny observed in some of the crosses were small, they all illustrated single gene inheritance and absence of sex-linkage. Nunoya et al. (1983) did not give a descriptive name to the mutant nor did they assign a gene symbol. Because of its similarity to the arched neck syndrome of Conner and Shaffner (1953), and because it is a convulsive disorder, it is suggested here that the trait be called arched neck seizures and that the gene symbol *ans* be adopted.

The mutation described by Nunoya et al. (1983) has been of particular interest to the author, and to Smyth (1989, personal communication), because of the occurrence of a similar syndrome in North American Fayoumi stocks. The author's Fayoumis were derived in 1964 from those of Smyth, whose flock traced to the original importation of Fayoumis from Egypt to Iowa State University in 1948. Seizures like those described by Nunoya et al. (1983) have been seen repeatedly in the author's stock, but they have not been investigated genetically. Seizures in Smyth's birds were first thought to be similar to those of the arched neck syndrome (Conner and Shaffner, 1953), but may have actually been more like those found by Nunoya et al. (1983); genetic investigation of them was attempted but it was discontinued because of difficulty in identifying affected birds and in stimulating seizures for observation. There is strong likelihood that the Japanese Fayoumi stock also traces to the 1948 American importation from Egypt, in which case the syndrome seen in three different flocks may be result of a single mutational event.

Paroxysm (*px*). Discovery of the paroxysm mutant described by Cole (1961) should perhaps be credited to a dutiful but superstitious animal care attendant, who was charged with care of a newly hatched group of crossbred chicks. All went well for a few weeks. But then he began finding birds in what appeared to be violent and final death throes; they thrashed about and then lay still. He carefully placed them by the pen door for later delivery to the necropsy room, and went about servicing the pen. But when he returned to the door, the dead birds were gone. After several such events he became badly frightened and appealed for help. A compassionate scientist went with him. Upon entering the pen they soon saw a bird undergoing severe convulsions terminating in a coma. But after several minutes the coma ended, the bird regained its feet and rejoined its flock mates. The attendant's peace of mind was restored, and the paroxysm mutant was revealed.

Paroxysm first appeared among female progeny of a Houdan x White Leghorn male mated with Ancona females (Cole, 1961). Seizures began at about two weeks of age following auditory or visual stimulation. The seizure consisted of rigid extension of the legs, with head thrown back, wings beating violently, and the entire body in tetany and tremors. Then the body became completely relaxed, with the eyes closed; complete prostration was of variable duration. When the bird recovered, it walked for a while on its toes, taking quick short steps; to assist in maintaining balance, the tail would be spread like that of a strutting turkey. The seizure could be repeated after a short interval of recuperation. Growth rate was very poor, and all affected individuals died by 15 weeks of age.

Since the condition occurred in about half of the daughters of a carrier sire, and never in his sons, it was evident that the trait was sex-linked and recessive (Cole, 1961). The condition was named paroxysm and the gene symbol *px* was assigned. Because all affected females die before sexual maturity, the mutant can be maintained only in heterozygous males. It has not been possible to produce males that are homozygous for the condition, so that its expression in that sex is not known.

Paroxysm has subsequently been investigated intensively by Kuenzel and by Beck (Schaefer) in association with their students and colleagues. Their motivation has been the potential use of the paroxysm syndrome in studies of central nervous system excitability, and as an animal genetic model of human grand mal epilepsy.

Kuenzel and Rubenstein (1974) reported an important series of basic experiments to measure behavioral and physiological parameters of *px* chicks. They found that the average age at first seizure was 19 days, with a range of 9-29 days. The onset of seizures was accompanied by anorexia and progressive wasting of body fat and protein stores. Average lifespan was only 36 days. The seizure syndrome could be divided into four phases: alert phase (vocalization and/or running — 1-2 seconds), clonic phase (wing flapping, leg flexing — 1-2 seconds), tonic phase (opisthotonos, legs extended — 13 seconds), and recovery phase (coma — 34 minutes). They found that seizures could be precipitated by any external stimulus that startles the chick, including auditory, visual, tactile, and photostimulation. Auditory stimulation was the most effective and photostimulation was the least, hence the seizures are usually described as audiogenic. Electrical stimulation of the hyperstriatum was consistently effective in stimulating seizures, and it was useful in studies of the electroencephalogram. Subsequently, Rhody and Kuenzel (1981) used electroshock to quantitate the effects of seizures on blood pressure, heart rate, respiratory rate, and the electroencephalogram.

One of the problems in working with the mutant has been that of identifying *px* individuals before nine days of age when the seizures first appear and the anorexia begins. Schaefer and Kuenzel (1978) found that by drawing a large volume of blood through heart puncture, seizures are invariably induced in *px* chicks regardless of their age; this has been a very useful but drastic indicator of genotype in very young birds. Cole (1961) had found that the complete seizure pattern was produced immediately following cervical dislocation. Thus far no marker gene on the Z chromosome has been found which could be linked to *px* for easy identification of paroxysmal chicks at hatching.

Although Cole (1961) and Kuenzel and Rubenstein (1974) were unable to find any gross or microscopic abnormalities in any region of the brain or spinal cord, Beck et al. (1983) were able to demonstrate that nuclei and fibre tracts of the auditory and vestibular systems were extensively degenerated. Deep nuclei of the cerebellum appeared to be seriously affected by five days of age, and maximum degeneration of all components of the two sensory systems was evident by 20 days of age. However, for reasons not yet fully explained, auditory stimulation continues to elicit seizures throughout the life of the chick, indicating auditory function despite extensive cell death (Beck et al., 1987).

The recent work of Beck and her associates has mostly concerned the mechanisms underlying the seizures, neural degeneration, and anorexia. Thus far none has been demonstrated to be the exclusive cause, but evidence has accumulated which implicates abnormal oxidative metabolism. It was shown that noradrenaline levels in the cerebellum of *px* chicks were low throughout the birds' lifespan (Schaefer and Kuenzel, 1978); since the cerebellum is the moderator and coordinator of voluntary movement, this finding may explain the motor excitability of *px* chicks. There is a strong indication of abnormal iron metabolism, which may be associated with degeneration of brain areas that are vulnerable to hypoxia (Beck and Kuenzel, 1983). Levels of γ-aminobutyric acid (GABA) are elevated in *px* chicks (Firman and Beck, 1984); since GABA is a major inhibitory neurotransmitter and energy metabolite, and is strongly implicated in epilepsies, the abnormal levels in paroxysmal chicks may be a causative factor in seizure incidence. Brain energy reserves may also be involved (Firman and Beck, 1985); the chick functions normally until 7-9 days of age using the yolk sac as a primary energy source; but by ten days of age, brain levels of adenosine triphosphate and phosphocreatine are both reduced, indicating that replenishment of energy reserves is not occurring, which may be explained by a failure to switch from yolk sac lipids to dietary carbohydrates as an energy source.

Epileptiform seizures (*epi*). Credit for discovery of the epileptiform seizures mutant should also go to a poultryman. Fayoumi eggs had been acquired from far away for behavior genetics research. At hatching, several chicks were culled by the scientist, presuming them to be severe cases of congenital loco, but all the others appeared to be normal. The caretaker was cautioned about the wildness of Fayoumis, and he duly reported each day on how very wild they were, saying that when they were frightened they buried themselves in the wood shavings litter just like wild partridges. Suspecting a practical joke, to which both caretaker and scientist were prone, an investigation ensued revealing five chicks that exhibited severe convulsions when disturbed and that did indeed become litter-covered during the seizures; all of the others were typically nervous and flighty Fayoumis but not at all like 'wild partridges'.

Subsequently the mutant has become a valuable animal model of grand mal epilepsy. Of particular relevance are its single gene inheritance, and its brainwave patterns and drug responses which have close similarity to those of humans with grand mal epilepsy. There is an extensive literature concerning the mutant. Detailed reviews of genetics and behavior, the electroencephalogram, drug responses, and brain biochemistry are provided in Ookawa's (1983) *The Brain and Behavior of the Fowl.*

The mutant was discovered among Fayoumi chicks obtained as hatching eggs from the University of Massachusetts (Crawford, 1970). Presumably it had arisen in the parent flock but it was never witnessed there. It was established in a synthetic stock with a very broad genetic base, where it has been maintained. All published reports have involved that synthetic population. Subsequently it was returned to the pure Fayoumi genome through repeated backcrossing. The seizures in that genetic background are more severe than in the synthetic line, and this may have value in future comparative studies.

A general description of the seizures was reported by Crawford (1970) and a more detailed characterization was given by Crichlow and Crawford (1974). Seizures can occur throughout the lifespan of the bird. They first are seen when day-old chicks are removed from the incubator-hatcher. Incidence and severity decline greatly from 3-7 days of age, and then return to nearly total expression, where they remain into old age (Lee, 1973). There are three phases in the seizure pattern (Crichlow and Crawford, 1974). Phase I (5-7 sec) is indicated by increased alertness, flattened feather coat, excited vocalization, and an upward and backward extension of the neck; the bird makes pecking motions laterally and towards the dorsum. Phase II (5 sec) is indicated by loss of leg muscle control; the bird staggers, sits on its hocks, and may extend its wings for support. Phase III (30-35 sec) consists of violent extension and flexion of the legs and wings; the bird tries to run; it tumbles and thrashes uncontrollably. The bird then collapses in a coma, which may persist for several minutes. When it regains its feet, it assumes a roosting attitude and is unresponsive to external stimuli; it may remain stationary for a prolonged period. The full seizure pattern can be repeated after a brief interval of recuperation (Lee, 1973).

Seizures can be induced by a variety of stimuli. Crawford (1970) described some of these, concluding that 'nervous fatigue' was an element common to them all. Lee (1973) found that heat stress was also an inducer. Crichlow and Crawford (1974) showed that the most effective stimulus under laboratory conditions was intermittent light stimulation (ILS) from a strobe lamp. Lee (1973) made a detailed study of the response to ILS in relation to flash frequency, age, and sex of subject. He found that the most effective ILS frequency at all ages and in both sexes was 10-25 flashes per second (fps); the mode was 14 fps. ILS stimulation at 14 fps has since been used in all experiments and in identifying genotypes of breeding stock. Nearly all affected individuals will develop a seizure within 20 sec of ILS exposure.

Observation of breeding records, and results from breeding tests, have indicated that epileptiforms seizures is caused by an autosomal recessive gene (Crawford, 1970). The gene symbol *epi* was assigned to it. Segregation data obtained using crude methods of seizure induction indicated that penetrance of the gene was incomplete. However, when genotypes were identified using ILS stimulation under controlled conditions, penetrance was found to be complete (Crawford, 1983).

Detailed studies of brainwave patterns in the *epi* mutant were reported by Crichlow and Crawford (1974) and by Crichlow (1983). The main distinguishing feature between epileptic and normal chickens is in the resting electroencephalogram (EEG). It is characterized in epileptics by high-amplitude slow waves which are not present in the EEG of normal chickens. Bilateral spiking appears preceding light-induced convulsions, and is reminiscent of similar spiking which occurs in humans before grand mal convulsions. The ILS frequencies which induce seizures in chickens are within the range which is stimulatory to humans having photosensitive epilepsy.

Most research attention has been given to studies of drug responses and brain biochemistry, in relation to the *epi* mutant serving as an animal model for the human disease. It has been of particular interest to determine whether the *epi* mutant is a good *pharmacological* model of grand mal epilepsy, that is, if it will respond to the same drugs and at the same plasma concentrations that are effective in humans. Cumulative research results were summarized by Johnson and Davis (1983). It has been found that seizures in *epi* mutant chickens can be attenuated or abolished by phenobarbital, phenytoin, and primidone at plasma concentrations which are effective in human patients. People suffering from petit mal seizures can be protected by ethosuximide; it is ineffective in *epi* chicks. Valproic acid, which is effective against many seizure types in humans, and trimethadione, which is effective against petit mal but not against grand mal, both protect mutants against seizures. They are also protected by diazepam and clonazepam.

Based on this evidence that the *epi* mutant is an acceptable pharmacological model of the human disease, research has been conducted on new or proposed anticonvulsant agents for use in human patients. Johnson et al. (1975) demonstrated that incidence and severity of seizures induced by IPS could be reduced by Δ^9-tetrahydrocannabinol, and that abnormalities in the EEG could likewise be reduced. Crichlow et al. (1986) found that *epi* mutant chickens could be protected against seizures by ivermectin, an antiparasitic agent that acts against the γ-aminobutyric acid (GABA) system of the parasite.

It has also become possible to use the *epi* mutant as a milieu for studying the mechanism of pharmacological and biochemical action of drugs. For instance, a series of reports has concerned the interaction of benzodiazepines with specific high-affinity binding sites in exerting their effects (see Pedder et al., 1987 for review).

The fortuitous finding by Lee (1973) that seizures could be induced in *epi* mutant chickens by hyperthermia, has led to investigations which may have relevance to febrile seizures in children. There is no fully satisfactory animal genetic model for that disease. Johnson et al. (1983) verified that febrile seizures could be produced in chicks but not in adult birds, and then demonstrated that phenobarbital, which is protective in children, delayed the onset of seizures in *epi* chicks; neither valproate, which is effective in humans, nor phenytoin, which is not, gave any relief to hyperthermic chicks. Subsequently it was shown that the latency to onset of seizures could be increased by elevating the concentration of GABA in the brain (Johnson et al., 1985; Pedder et al., 1988); similar findings pertain to children with febrile seizures.

Throughout these investigations it has been of interest to seek causes or changes associated with the epilepsy syndrome. Despite prolonged searching, no microscopic lesions or defects have been found. Johnson and Davis (1983) have reported that cerebral hemispheres from epileptics are heavier than those from normal birds, reflecting a higher protein content and not a difference in water content. Most interest has centred on biochemical and neurochemical differences. Johnson and Davis (1983) have shown that cerebral hemispheres in *epi* mutants have elevated noradrenaline concentrations, and low dopamine and 5-hydroxytryptamine concentrations, but these appear not to be involved directly in high seizure susceptibility. They have also detected abnormalities in the cholinergic enzymes acetylcholinesterase and cholineacetyltransferase, and have demonstrated a deficiency of endogenous phosphorylation of at least three brain proteins, but the functional significance of these differences is not known. Crawford et al. (1984) have shown that epileptic and normal chicks do not differ in levels of cyclic nucleotides, which are important in normal functioning of neuronal tissue. The studies of Crichlow (1983) have concerned the role of brain GABA in determining seizure susceptibility. He has shown that the level of GABA in whole brain tissue of *epi* mutants is lower than in normal birds, and that it is reduced further during IPS-induced seizures. He has also shown that the deficiency in GABA is not related to alterations in activity of the enzymes which synthesize and degrade GABA. Further support for an involvement of brain GABA has come from the finding that the antiparasitic agent ivermectin, which enhances GABA release, is effective in protecting against seizures (Crichlow et al., 1986).

NEUROMUSCULAR VARIANT

Tipsy (*tip*). The tipsy mutant differs considerably from all of the other nervous system variants. A brief description of the condition was provided by Hawkes (oka Hawes) et al. (1973). It was suspected that the primary defect was neural, but detailed studies were not reported. The condition was not evident at hatching; the average age at positive identification was 51.3 days. There was a progressive decrease in the ability of affected birds to maintain a steady gait. Severity of crippling was variable. Those worst affected died by three months of age. Others survived to maturity if given extra care, and they were able to reproduce. Severely crippled individuals exhibited marked atrophy of the muscles of the pelvic girdle and the pelvic limb.

Tipsy was found in Partridge Wyandotte chicks. It was reproduced by mating affected females to their normal sire. When presumed carrier males were mated to White Leghorn females, all of the F_1 progeny were normal. In the F_2 generation from eight pair matings there were 94 normal and 22 crippled chicks, including both males and females. It was concluded that the condition was caused by an autosomal recessive mutation. It was given the name tipsy and the gene symbol *tip*.

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Chapter 10. MUTATIONS AND MAJOR VARIANTS OF OTHER BODY SYSTEMS IN CHICKENS

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In the five preceding chapters, special consideration has been given to mutations affecting particular parts or systems of the body in chickens — color of the plumage and skin, morphology of feathers and skin coat, biochemical variants, and genetic abnormalities of the musculo-skeletal system and of the nervous system. A few mutations have been studied which affect several other body systems in chickens — the respiratory, reproductive, urinary, hormonal, and circulatory systems, vocal performance, and the eye. These are considered together in this chapter of miscellanies.

RESPIRATORY SYSTEM

Gasper (*ga*). Only one mutation which affects the respiratory system has been reported in the technical literature. Price et al. (1966) provided a brief description of a respiratory abnormality found in an inbred meat-type strain of chickens. Soon after hatching, affected chicks exhibited a characteristic 'snick', a bronchial rattle, and a characteristic gasping. The condition was most severe by four days of age and mortality was very high. Those surviving to adulthood continued to have a bronchial rattle and they did not make normal weight gains. No infectious agent could be isolated nor could evidence of inflammatory processes be found. When affected males were mated to unrelated normal females, it was found that none of the sons had the defect, but about half of the daughters were affected. These observations indicated that the abnormality behaved as a facultative lethal and was caused by a sex-linked recessive gene. The name gasper and the gene symbol *ga* were assigned.

The mutant was provided to Macdonald College of McGill University where it was investigated by D. McKay, an undergraduate student (Hawes, 1975, personal communication). Oxygen consumption was found to be greater in gaspers than in controls, and gaspers exhibited greater depth of breathing. It was postulated that an allergic phenomenon might be involved.

The mutant was transferred to University of Saskatchewan, where expression of the abnormality became progressively weaker with each generation until it was no longer evident and the mutant line was discarded. A sample of the line sent to Nova Scotia Agricultural College suffered a similar fate.

REPRODUCTIVE SYSTEM

Several genetic variants of the reproductive system in chickens have been described in the technical literature. One of them, hereditary gonadal hypoplasia (*Hgh*), affects only males. Another, atresia isthmi (*Aro*), changes reproductive tract anatomy in both males and females. The condition described as right oviduct development (*Rov*) is expressed only in females. There may be more than one genetic cause for this anomaly. Two variants affect function of the female reproductive tract. Restricted ovulator (*ro*) prevents normal development and release of ova. Riboflavinuria (*rd*) affects deposition of riboflavin in the albumen; it is described in Chapter 32.

Hereditary gonadal hypoplasia (*Hgh*). Lojda and Hovorka (1968) reported low sperm concentration with a relatively high percentage of pathological spermatozoa in semen from a White Leghorn male having testicular hypoplasia. Fertility of that male was significantly lower than that produced by a normal male sib. Incomplete dominance was proposed as the mode of inheritance. Subsequently, fertility levels obtained in test matings of daughters supported the suggestion that the condition was incompletely dominant (Lojda and Hovorka, 1969). Somes (1980) assigned the gene symbol *Hgh* to the condition.

Atresia isthmi (*Aro*). A hereditary closure of the oviduct in White Leghorns was described by Finne and Vike (1951). Affected females were unable to lay eggs because of rupture of the oviduct at the isthmus. Rupture at the isthmus was frequently accompanied by a constriction between the vagina and the cloaca. The ovary was normally developed, and nesting behavior was strongly evident, but soon after sexual maturity the birds developed signs of internal laying and subsequent mortality was high. At necropsy, the imported sire of affected females from the first generation was found to have a discontinuous right vas deferens, and the right testis was flabby and wrinkled. Further generations of affected females were produced, and the defect was also transmitted in a cross with Jaer-hens from a strain kept closed for several decades. The condition thus appeared to be controlled by a single dominant gene. But since affected females could not produce eggs it was never possible to determine whether the gene was autosomal or sex-linked, nor was it possible to generate homozygous affected males for further study. Somes (1980) assigned the gene symbol *Aro* to the condition, referring to it as atresia of reproductive organs.

Right oviduct development (*Rov*). There are many reports in the literature about hens with right oviducts (see reviews by McBride, 1962, and by Sell, 1959). In the normal situation, only the left oviduct is retained as a functional organ (Chapter 3); the right is a nonfunctional vestige attached to the cloaca, and only in abnormal situations does it become enlarged and filled with fluid. McBride (1962) placed reported cases into four classes:

- right and left ovaries and oviducts fully developed
- right oviduct developed with some gonad development on the right side
- right and left oviducts developed but complete absence of a right gonad
- left oviduct and ovary normal with an imperfect (usually cystic) right oviduct.

The last two classes include most of the reported cases. It is significant that there are no reports in the literature of a hen having a reproductive system on the right side and none on the left.

At least some of the reports of persistent right oviducts indicate that a genetic basis is involved, as for instance the high incidence reported by Sell (1959) in two generations of a White Plymouth Rock strain. Two other strains are of particular interest in this regard. One was a Black Australorp strain in Australia, and the other an inbred line of Rhode Island Reds in the United States.

The Rhode Island Reds described by Morgan and Greb (1959) had an inbreeding coefficient of 75-80 percent. The noninbred stock from which they had been derived was available for comparison. A sample of 52 hens representing four generations of the inbreds was found to have 90 percent incidence of persistent right oviducts, compared with 42 percent in the control sample. Furthermore, over 60 percent of the right oviducts from the inbreds were greater than half the length of the normal left oviduct, but those of the control sample were all very short. A decade later the inbred line was placed with another laboratory where it was maintained for at least eight further generations without selection for the right oviduct trait. At that time observations were made on 82 hens (Wentworth and McGibbon, 1980). Complete left and right oviducts were found in 74 percent of the birds, in ten percent the right oviduct was nonfunctional due to a constriction at the isthmus-magnum junction, 22 percent had an incomplete right oviduct, and four percent had only the normal left oviduct. There is no doubt that duplication of oviducts in this inbred line of Rhode Island Reds is under genetic control, but there has been no demonstration that the control is monogenic.

The Australian strain was also inbred, and it had a long history of selection at one location (McBride, 1962; Winter, 1958). Soon after discovering the anomalous double oviducts in the strain, 133 adult hens were examined and classified. The strain was subsequently divided into three lines for other purposes, and further observations on 264 hens were taken six years later. All of these data were tested against the hypothesis of genetic control by a single incompletely dominant autosomal gene, in which *Ro*/*Ro* represented birds with a fully developed right oviduct which was patent throughout, Ro/ro^+ individuals had a partially developed and usually cystic right oviduct, and ro^+/ro^+ birds were normal. Observed and expected ratios were in remarkable agreement. Unfortunately the *Ro* gene symbol was also used to described the restricted ovulator mutant. To avoid confusion between the two Somes (1981) recommended that McBride's (1962) right oviduct variant be designated as *Rov*, and the wild-type recessive allele as rov^+.

Restricted ovulator (*ro*). The restricted ovulator condition was first described to poultry scientists by Jones et al. (1975). McGibbon (1977) showed that the causative gene is sex-linked and recessive, and that it is linked to k^+ and id^+ loci.

Affected hens lay few or no eggs. The occasional eggs are smaller than those of normal hens, and their yolks are translucent and deep orange in color as compared with normal yolks which are opaque and yellow. Affected hens exhibit extreme hyperlipemia (Ho et al., 1974), and they fail to incorporate into the yolk the normal amounts and proportions of low density lipoprotein and lipovitellin (Schjeide et al., 1976). Birrenkott et al. (1975) found higher than normal serum levels of luteinizing hormone and estradiol, but normal levels of progesterone. Grau et al. (1979) showed that yolks of eggs laid by affected hens contain less iron and copper, and more sodium and potassium, than those of controls. They suggested that the *ro* gene may interfere with normal iron absorption, metabolism, or utilization leading to both hyperlipemia and a low level of iron in the yolk. They further postulated that iron metabolism may indeed be central to explanation and understanding of the condition.

URINARY SYSTEM

Five genetic studies have been reported that involve the chicken urinary system. Two involved hypoplasia of the kidney and three concerned the susceptibility of chickens to articular gout and its associated plasma uricemia.

Kidney hypoplasia (*kh*). Kidney hypoplasia in the chicken has been studied by two groups of investigators. Jeffrey et al. (1937) found in a strain of White Leghorns a condition in which the left kidney was absent or atrophied. In four generations, 14 affected females were found, all descended from two males. Although no genetic ratios were evident, the condition was suspected to have been inherited. Because only females were affected, the possibility that it might have been a sex-limited trait was suggested. Since most males were discarded early in life and were not examined, the genetic basis of this condition is still in question. No apparent ill effects were experienced by abnormal birds.

Pun (1961) studied a kidney hypoplasia condition in Brown Leghorns that appeared to be due to a simple autosomal recessive. The gene showed variable penetrance and expressivity, with a greater expression being shown in females than in males. There was an anteroposterior gradient in the frequency distribution of the defect among the kidney divisions, but no difference in frequency between the left and right sides. Compensatory hypertrophy was observed only in cases of unilateral hypoplasia and then only when all three divisions were affected. Reciprocal crosses between heterozygous carriers and normal birds produced all normal chicks, while from five F_2 matings 195 normal and 71 affected offspring were produced. The offspring from all five matings segregated in a 3:1 ratio except for the female progeny from one dam. Except for this one case, the data fitted the hypothesis that this condition was due to a single autosomal recessive gene. No gene symbol was assigned by Pun (1961), but the gene symbol *kh* has since been assigned to this trait (Somes, 1980).

It is impossible to determine whether these two kidney hypoplastic conditions (Jeffrey et al., 1937; Pun, 1961) are related. The condition studied by Pun (1961) readily segregated, affecting both sexes and both kidneys, while that of Jeffrey et al. (1937) did not give ratios and it affected only the left kidney of females. It would appear that although both have a genetic basis, different factors are probably involved.

Hereditary articular gout and uricemia (*go*). Articular gout may occasionally occur in birds when diets are unusually high in protein. Tophi develop on the feet and hock joints, and plasma uric acid levels are elevated. Three studies, in which susceptible and resistant chicken lines were developed while the birds were being fed high protein diets, demonstrated that the development of articular gout and its associated hyperuricemia has a genetic basis.

Peterson et al. (1971) observed an incidence of 38 percent articular gout when an experimental line of chickens was fed a diet containing 80 percent protein. Divergent selection was carried out for three generations with the result that the high line progeny exhibited 100.0, 100.0 and 98.7 percent gout, while the low line exhibited only 33.3, 28.8 and 27.7 percent gout for each of the generations. These data were taken as evidence that susceptibility to developing gout while on a high protein diet was a recessive genetic trait. Plasma uric acid levels were elevated in the susceptible line when compared to the resistant line. This hyperuricemic condition of the high line was even evident when comparison was made on a low protein diet.

Komiyama et al. (1977a) examined three strains of chickens for their susceptibility to diet induced articular gout. A Fayoumi and two White Leghorn strains were fed diets consisting of either 20, 40 or 70 percent protein. No gout was evident in either White Leghorn strain. The Fayoumi strain showed 25.9 percent gout on the 40 percent protein diet and 47.8 percent gout on the 70 percent protein diet. It was then divergently selected for incidence of gout while on a 40 percent protein diet. By the second generation of selection, the gout line showed 100 percent gout while the nongout line showed only 5.8 percent incidence. Reciprocal crosses between the lines showed that gout susceptibility was due to a single autosomal recessive gene (Komiyama et al., 1977b), which later was assigned the gene symbol *go* (Somes, 1980). Hirai et al. (1977, 1978) observed an increase in urate excretion in these gouty males when maintained on the 40 percent protein diet, and they attributed this to accelerated purine and urate biosynthesis.

Two uricemic-articular gout lines were developed over a period of ten generations of selection at Cornell University (Cole and Austic, 1980). The high uric acid line (HUA) had greater than threefold increases in its plasma uric acid levels when compared with the low uric acid line (LUA). Selection was based on plasma levels of uric acid while the birds were on a 20 percent protein diet. By as early as the fourth generation of selection it was clear that two lines had differentiated. It was evident that by selecting for increased plasma uric acid levels even while on a lower protein diet they were at the same time selecting for increased susceptibility to diet induced articular gout. This agrees with the observations made by Peterson et al. (1971). Because of the rapid progress made during selection and the rather rapid plateauing effect encountered, it was suggested that very few major genes were involved.

Both the color gene (i^+) and the dominant white gene (I) were segregating in these lines. Comparisons between colored and white birds showed that the colored birds (i^+/i^+) had significantly higher blood uric acid levels than did the whiles (I/I or I/i^+). The original stock from which these lines were developed were New Hampshires (i^+/i^+) with White Leghorns (I/I) being introduced in subsequent generations. It was suggested from these data that a major recessive gene for elevated uric acid levels was probably linked with the i^+ locus (Cole and Austic, 1980).

Several studies have been undertaken to try to determine the metabolic basis of inherited hyperuricemia. Using standard clearance procedures, it was shown that the uricemia and gout of the HUA line was not due to excessive synthesis of uric acid but to impaired renal clearance of uric acid (Austic and Cole, 1972). Further work (Zumda and Quebbemann, 1975) localized the defect to a transport mechanism in the kidney peritubular membrane. Specificity of the renal transport impairment was assessed and shown to be specific to organic acids (Austic and Cole, 1974).

It is quite evident that all three of these uricemia-articular gout conditions in the chicken have a genetic basis. It is not clear, however, as to whether any or all of them are the same. They all produced gout when placed on a high protein diet and all seemed to be inherited as recessives. The condition in the Fayoumi was reported as due to a single gene (Komiyama et al., 1977b). The diet induced gout lines of Peterson et al. (1971) showed more gout in the nongout line than was expected. This may have been caused by modifiers which prevented full expression of the trait, thus allowing susceptible birds to be used as breeders in the nongout line by mistake. The Cornell HUA line seemed to have at least one major recessive gene associated with it. Possibly other genes were involved, but reciprocal breeding tests were not reported (Cole and Austic, 1980). It is possible that one common recessive gene contributed to all three of these conditions, but this cannot be verified. Also it must be pointed out that the defect in the Cornell HUA line appeared to be different than that reported in the Fayoumi breed.

HORMONAL SYSTEM

Several studies have been undertaken to study genetic variations involving the thyroid gland and the pituitary-kidney water reabsorption system. Thyroid gland studies have centred around variations in sensitivity to thyrotropin and several hypothyroidal conditions.

Studies involving pituitary and kidneys have concerned the diabetes insipidus trait.

Thyrotropin sensitivity (*Tsh, tsh*). Munro et al. (1943) tested five commercial breeds of chickens for their sensitivity of response to a concentrated anterior pituitary extract which exhibited both thyrotropic and gonadotropic properties. Using an increase in thyroid gland weight as their response indicator, they were able to show differences among the breeds, with both sexes of the White Wyandotte breed having the greatest thyroid gland size increase and thus the lowest response threshold. Breed differences in thyrotropin sensitivity seemed to be indicated, and thus there was a suggestion of genetic control for this trait.

Premachandra et al. (1958) reported significantly different thyroxine secretion rates in two New Hampshire chicken lines that had been selected by El-Ibiary and Shaffner (1950) on the basis of their differential response to the goitrogen thiouracil. Progeny from matings of birds with low secretion rates were themselves low secretors, and progeny from high secretion rate matings had high secretion rates. When the parents were intermediate, their progeny had a graded range of secretion rates. High heritability estimates indicated the involvement of very few genes (Stahl et al., 1962).

Fodor et al. (1966) observed that medium-sized breeds of chickens seemed to be less sensitive to exogenous thyrotropin than bantams, and Fodor and Pethes (1974) demonstrated a monogenic basis for this difference. In crosses between Naked Neck Transylvanians and bantams (breed not specified) they were able to show that the higher degree of thyrotropin sensitivity characteristic of the bantam was determined by a single dominant allele. A recessive allele at the same locus was carried by the Naked Neck Transylvanian breed which resulted in its lesser degree of sensitivity. The genetic nature of this trait was independent of body weight and its phenotypic expression was not greatly affected by a goitrogen (methimazole), season, or sex. The gene symbols *Tsh* for the dominant allele and *tsh* for the recessive allele were later assigned (Somes, 1980). Which of these alleles is the wild-type cannot be determined from this study.

Bowen and Washburn (1982) also showed genetic variation in thyrotropin sensitivity in the Athens-Canadian Randombred broiler chicken line by using a bioassay of circulating thyroxin (T4) as an indication of response to injections of TSH. Using 30 sire families and assaying 833 chicks, they reported heritability estimates for T4 levels of 0.63 based on the sire component of total variance and 0.53 based on the dam component of total variance.

Each of these studies, in somewhat different fashion, has shown that sensitivity to TSH is under genetic control. Whether each was dealing with the same locus is impossible to determine. A single locus was identified in one case (Fodor and Pethes, 1974), but the other studies would also seem to indicate that not many genes were involved.

Recessive dwarfism (*td*). This particular type of dwarfism was first reported by Landauer (1929) in a single bird in a Rhode Island Red flock in Maine. The bird was small and the feathers were longer than normal. The thyroid gland was enlarged and consisted of aplastic tissue without colloid. This hypothyroidal condition closely resembled the human condition known as *myxoedema infantilis*. Although his description of the condition was extremely detailed, Landauer gave no genetic data.

Upp (1932) and Mayhew and Upp (1932) also reported a hypothyroidal dwarf condition in Rhode Island Reds, the description of which conformed to that of Landauer (1929). Chicks appeared normal at hatch, but decisive differences began to appear by three weeks of age. Legs were much shorter in proportion to body size, and the outer toes turned outward and backward. Their heads were wider in the eye region and their beaks were parrot-like. Their tails were carried on a level with the middle of the body with tail feathers directed backward and downward. Chicks were sociable and active, but difficult to raise to maturity. No dwarfs ever became sexually mature. From the pedigree record data, Mayhew and Upp (1932) suggested that a single recessive gene was the cause of this condition. Later, Upp (1934) conclusively showed that this type of dwarfism resulted from homozygosity for a recessive autosomal gene when his results combined with unreported data from Warren gave a 428:120 ratio, where a 411:137 ratio would be expected from carrier x carrier matings. Hutt (1949) later proposed the gene symbol *td* (thyrogenous dwarfism) for this gene.

Hereditary autoimmune thyroiditis (obese). The obese strain (OS) of White Leghorn chickens described by Cole (1966) was developed at Cornell University from 20 obese females which segregated out of the Cornell C strain during the 1955-57 hatching seasons. Through selection the incidence of the obese trait was increased from zero to over 80 percent in males and from less than one percent to over 90 percent in females (Cole et al., 1968). Expression of the obese trait was somewhat more frequent and more pronounced in females than in males. Obesity was the first observed characteristic of these birds and thus the original name for the selected strain. Obese chickens were generally recognized by 6-10 weeks of age as having features typically associated with hypothyroidism. They were smaller in size, with soft plump pliable skin and large accumulations of fat. Feather structure was silky and excessively long. They had quiet dispositions and were sensitive to low temperatures. Sexual maturity was delayed and egg production was poor, although egg size was normal. The addition of protamone to the diet greatly improved production. Fertility and hatchability were generally normal (Cole, 1966).

The autoimmune nature of this condition was first documented by Cole et al. (1968) and the trait is now generally referred to as spontaneous autoimmune thyroiditis (SAT). It is used extensively as a model for Hashimoto's disease, a human chronic thyroiditis. Wick et al. (1982) have reviewed the clinical histopathological and serological characteristics of the OS birds and compared them with Hashimoto's disease. During the 22 years since its discovery, the OS chicken has been used in hundreds of studies and a vast literature exists concerning this trait. A detailed coverage of this work cannot be presented here, but the reader is referred to reviews by Wick et al. (1985, 1986) for information on the immunopathology of SAT.

From his early crosses, Cole (1966) determined that SAT was inherited as a polygenic trait which was not completely recessive. Twenty-two years later the genetics of SAT is still not completely known. The latest theory (Hala, 1988) proposes that about five major genes regulate the full expression of SAT. Two gene families are suggested in which two genes of one family encode for the abnormal reactivity of the immune system, and three genes of the second family, one of them recessive, encode for the target organ susceptibility to the autoimmune attack. Minor modifying factors are also involved. These include such things as non-MHC immune response genes, sex hormones, and the effect that iodine level in the food has on thyroid susceptibility.

Diet-induced thyroiditis. The Cornell C strain (CS) of chickens showed a surprisingly high natural incidence of thyroid hormone (T3 and T4) and thyroglobulin autoantibodies (Brown et al., 1985). About 29 percent of CS females had antibodies to T3, 18 percent to T4, and 16 percent to thyroglobulin. The addition of iodine to the diet during the first ten weeks of life greatly increased the incidence of autoimmune thyroiditis in these birds, as determined by histological examination of the thyroid glands and measurements of autoantibodies, whereas an iodine-deficient diet resulted in a decrease in incidence of the disease (Bagchi et al., 1985). High levels of dietary iodine have been shown to increase the immunogenicity of the thyroglobulin molecule (Sundick et al., 1987).

The CS strain of White Leghorns is very susceptible to dietary iodine-induced autoimmune thyroiditis. Without a doubt this susceptibility is genetically based and probably multifactoral. The condition would appear to be different from that reported in the obese strain, as thyroglobulin autoantibody production in that strain was largely independent of dietary iodine intake (Sundick et al., 1987).

DAM line hypothyroidism. Smyth et al. (1981) reported that a low incidence of hypothyroidism was characteristic of their DAM line. This feature of the DAM line first appeared in the G_3 generation and its incidence had increased to 4.7 percent by the G_5 generation. These hypothyroidal birds closely resembled the Cornell Obese strain in appearance, responded to thyroid replacement therapy, had immune cell infiltration of the thyroid, and a complex multifactoral mode of inheritance. It is quite possible that these conditions are similar.

Diabetes insipidus (*di*). Buss and Murphy (1965) reported a strain of White Leghorns at Pennsylvania State University which was characterized as producing watery droppings (polyuria) and drinking excessive amounts of water (polydipsia). Examination of pedigree records suggested that the cause was probably genetic. Crosses subsequently showed that this condition was inherited as a single autosomal recessive (Dunson and Buss, 1968) to which the symbol *di* was assigned (Somes, 1980).

Chickens homozygous for diabetes insipidus (*di*/*di*) appeared to be normal in viability, fertility, feed consumption, body weight, egg numbers and weight, shell thickness, and albumen quality (Buss and Murphy, 1965; Buss, 1974). However, they showed a variable high degree of polyuria and polydipsia, which was expressed by three weeks of age with water intake about three times above normal and water/food ratios greater than two (Dunson and Buss, 1968; Dunson et al., 1972). Plasma osmotic pressure and sodium concentration were, however, very similar in normal and polydipsic birds.

Injections of aqueous lysine vasopressin or aqueous arginine vasotocin (Dunson and Buss, 1968; Raffel and Buss, 1975) showed that *di*/*di* birds are lysine vasopressin and arginine vasotocin sensitive and suggested that the lesion may affect the quantity of the hormones produced. However, in another group of experiments (Dunson et al., 1972), neither polydipsia nor polyuria were affected by injections of vasopressin. This observation, along with a series of studies on water restriction, saline solution consumption, and pituitary analyses, led these investigators to conclude that there is no blockage of arginine vasotocin synthesis. They suggested that the condition was a mild case of nephrogenic diabetes insipidus. The data in the study of Dunson et al. (1972) seem to be in conflict with the reports of Dunson and Buss (1968) and Raffel and Buss (1975). Thus two different causes for the condition have been suggested, with each ruling out the other. It is clear from these data that the condition has a simple genetic basis, but its primary cause is less clear.

A similar condition has been reported from France in the M99 and Jouy strains of chickens (Bordas et al., 1978). This polydipsia was caused by a single major recessive gene, presumably the same as *di*. The *di*/*di* birds had a water intake about double that of the Di^+/-birds. The investigators found that the residual feed consumption of polydipsic birds (after adjustment for body weight and egg production) was significantly higher than in normal birds, but that egg weight and egg numbers were similar. They also compared the performance of polydipsic and normal birds at a high environmental temperature (27-34°C cycle) to see if polydipsic birds were more resistant to heat stress (Obeidah et al., 1977).

At these temperatures polydipsic birds panted less, and layed significantly more and heavier eggs over the 8-day test period than did the normals.

CIRCULATORY SYSTEM

The circulatory system of the domestic chicken is quite resistant to viable genetic mutations. Only one genetic condition has been reported in this system.

Inherited anemia. Washburn and Smyth (1968) reported on the hematology of an inherited anemia which appeared simultaneously with a partial plumage achromatosis mutant. Their achromatosis-anemia stock was classified as macrocytic and normochromic. Erythrocytes from this line were greater in length, but not in width. The packed cell volume, hemoglobin content, and erythrocyte numbers of anemic birds were reduced by 15, 14, and 23 percent respectively. Data from genetic crosses suggested that the inheritance of this trait was multifactoral (Washburn, 1965) and that the achromatosis and anemia traits segregated independently of one another. A slightly normocytic, normochromic, spherocytic anemia with a seven percent reduction in packed cell volume was also observed in several gold columbian lines. Inasmuch as all these anemic stocks carried the *Co* gene, it was suggested that there might be a relationship between *Co* and reduced packed cell volume.

VOCAL SYSTEM

Extended crowing. During the era 206 B.C. - A.D. 1272 there were long-crowing fowl in China. Today in Japan there are four breeds, Tohtenko, Tohmaru, Minohiki and Koeyoski, which have long-crowing as a part of their breed characteristics. These are all related to the Shokoku breed, which is believed to be related to the Chinese long-crowing fowl (Oana, 1954). There is also a breed of fowl in South America known as the Brazilian Musician Fowl (Centola, 1978), and a German breed, the Bergische Kraher (Jeffrey, 1982), which have the same extended-crowing characteristic.

Birds with this trait are often called 'crow-at-dawn' birds, long crowers, extended crowers, musicians and singers. Typically a long-crowing male can crow for 10-20 sec whereas the length of a normal crow is 2-4 sec (Sasaki and Yamaguchi, 1970; Tengco and Nishimura, 1982). The record champion male of the Tohtenko breed crowed nonstop for 24 sec, while for the Tohmaru breed it was 25 sec (Tengco and Nishimura, 1982). There has been no genetic analysis of this trait, but because it is a breed characteristic it is undoubtedly under genetic control.

MUTATIONS INVOLVING THE EYE

The eye of the chicken shows the basic features found in all vertebrate eyes (Meyer, 1986). It has an internal sensory layer, the retina, a middle vascular layer, which includes the iris, ciliary body, and the choroid, and an external fibrous layer composed of the transparent cornea in the front and the cartilaginous sclera at the back. The eye is divided into two compartments by a biconvex lens suspended from the ciliary body by delicate ligaments: there are fluid filled aqueous chambers (anterior and posterior) between the cornea and the lens, and there is a large gelatinous vitreous body behind the lens. The aqueous humor that fills the chambers is responsible for intraocular pressure that maintains the shape and rigidity of the eyeball.

Functionally, the lens, which can alter its curvature, actively focuses images in the form of light rays on the retinal photoreceptors, the rods and cones. The rods and cones convert light energy into electrical potentials (nerve impulses) that are passed to the brain via the optic nerve. These electrical charges from the retina can be recorded by electroretinography (ERG). It is suspected that there may be a separate phototransduction process in the retina for regulation of diurnal changes in pigment migration and intraocular pressure.

The chicken eye also possesses some uniquely avian features. It is very large in relation to the size of the head compared to that of any nonavian vertebrates. The large size and flat shape permit the formation of a large retinal image. The chicken retina is also relatively thick compared to that of nonavian vertebrates, and has several specialized areas (foveas) for more acute vision. There are three types of visual photoreceptor cells in the retina: the rods, which are concerned with dim light vision, and the single and the double cones, for bright light vision and color discrimination. Specific functions of the double cones are still not clear. In chickens, double cones occupy about 82 percent of the retinal area (Matsusaka, 1963, as cited by Meyer, 1986). Like other birds, the chicken retina also has a pecten. The pecten arises at the exit of the optic nerve and projects into the vitreous body. It is highly vascularized and may serve as a nutritive organ, providing oxygen and nutrients for the inner portion of the retina.

Eleven genetic abnormalities affecting the ocular system of chickens have been reported. Five of these affect the size or shape of the eye, three cause retinal degeneration or dysplasia, and most of them result in blindness. The mode of inheritance has not been clearly demonstrated for all of the traits.

Bilateral microphthalmia (*mi, Mi-2,* and *mi-3*). Jeffrey (1941) reported a hereditary bilateral microphthalmia found in Barred Plymouth Rocks. The trait appeared to be inherited as an autosomal recessive. Affected birds were characterized by eyeballs with diameters approximately half that of normal birds. The eyeball was not visible on the exterior and there was a marked depression on each side of the head in the region of the eyes. The same mutation also affected the comb, which was thickened and sometimes doubled. The hatchability of affected embryos ranged from 25-43 percent and none of the chicks survived to maturity. Chick mortality was thought to be due to the birds being blind and unable to find feed and water, but lethality of the microphthalmic chicks is more likely to have been a pleiotropic effect. Somes (1980) assigned the gene symbol *mi* to this mutation.

Wight and Carr (1965) observed a normal Brown Leghorn hen producing progeny with unilateral or bilateral microphthalmia in two different periods when she was inseminated with semen from different and unrelated males. Of the 59 progeny produced, 18 (33 percent) were defective. The defects ranged from unilateral anophthalmia (no evidence of any eye structure on one side of the head) to eyes that were only slightly smaller than normal. There was a wide variation in the degree of abnormality in the eye structure of the affected chicks. None of the affected chicks survived to reproduce. Three female and one male normal progeny from this hen were bred but no microphthalmic chicks were recovered. Although the authors concluded that this was a dominant trait with incomplete penetrance (*Mi-2*), the evidence presented was not conclusive. Many cases of unilateral microphthalmia found in chickens and other animals are of nongenetic origin (Jeffrey, 1941).

More recently, Finzi and Romboli (1978) found another recessive microphthalmia (*mi-3*) in New Hampshires. This mutation seems to be different from the one reported by Jeffrey (1941). While not mentioned specifically by Finzi and Romboli (1978), it was implied that the microphthalmia was bilateral. As with the *mi* gene, the *mi-3* gene imposed a 30-45 percent prehatch mortality. Unlike the *mi* chicks, *mi-3* chicks were viable and adults could reproduce via artificial insemination. The expression of the *mi-3* gene was more variable than the *mi* gene. The eyes of some *mi-3* chicks approached normal while others were drastically reduced in size. The gene had no effect on the comb but affected the neck feathers. In affected males, feathers on the dorsal upper part of the neck were twisted along the shafts and gave the appearance of a little crest trailing along the back of the head. In females, the twisted feathers were found more on the sides of the neck. All microphthalmic birds had twisted neck feathers so it seemed to be a pleiotropic effect of the *mi-3* gene.

Somes (unpublished) found a hen with unilateral anophthalmia and made testcrosses among her normal sibs. The incidence of eye abnormalities among progeny of these matings ranged from 0-10.5 percent. The mating between the one-eyed female and a normal brother resulted in 17.2 percent of the progeny with eye abnormalities. Among all the progeny with eye abnormalities, 32 percent showed unilateral anophthalmia, 45 percent were affected bilaterally, and 23 percent showed various degrees of unilateral or bilateral microphthalmia. Almost all (98 percent) of the progeny with eye abnormalities were females. Unfortunately no further genetic tests have been carried out.

The frequency of microphthalmia in chickens and other animals is high but most cases are nongenetic in origin. Hereditary microphthalmia in chickens and in pigeons (Hollander, 1948) almost always involves lethality or semilethality, and none of the reported mutations has been maintained. A strain of chickens carrying a microphthalmia mutation may be useful for studying eye morphogenesis and importance of the eyeball as a mechanical factor in the development of its associated structures.

Pop-eye (*pop*). This Z-linked recessive mutation was first noticed in White Leghorns by the late W.H. McGibbon of the University of Wisconsin. Its phenotypic appearance consists of mild to severe bilateral protrusion of the cornea noticeable first at five weeks of age (Bitgood and Whitley, 1986), and thus the name 'pop-eye'. In adult birds, the marked protrusion of the cornea results in increased anterior chamber depth and subepithelial corneal scarring (Whitley et al., 1986). These alterations do not result in blindness but seem to cause astigmatism. In man, such an abnormality is known as keratoglobus. Histological studies of the pop-eye mutant revealed that there was a gradual degeneration of the cells of the corneal basal epithelium (Whitley et al., 1987). In adult pop-eye chickens, the corneal epithelium was decreased to two or three layers of flattened basal cells on an abnormal basement membrane and there was degeneration of the Bowman's layer. Garcia (1987, personal communication) also showed that cell types of the pop-eye corneal endothelium were different than those of normal chickens, and the age-related cell loss in this layer was significantly faster in the mutant compared to the normal. This noninflammatory dystrophic change in the corneal curvature due to central thinning and conical protrusion is known as keratoconus. The characteristics of keratoconus in the pop-eye chicken are very similar to those in man (Miller et al., 1988). Since the pop-eye mutation is the only known hereditary keratoconus in domestic animals, it may be very useful for studying the etiology and pathogenesis of keratoconus in man.

Blindness, enlarged globe (*beg*). This mutation was first observed by P. Hunton in a commercial strain of white-feathered brown-egg layers. Pollock et al. (1982) reported the mutation as being autosomal recessive but no data from testmatings were presented. The affected chicks were blind at hatch and overall size of their eyes was larger than in sighted controls. The mutant birds showed some degree of exophthalmos, exposing the inner canthus region of both eyes. Histologically a large number of small holes started to appear in the retina of *beg* embryos after eight days of incubation. These holes grew in size and number as incubation progressed so that by hatching many intercellular spaces were present in the retinas of affected embryos. The degeneration continued in the retina as the chicks grew, both photoreceptor cells and pigmented epithelium being affected. *In vitro* culture of cells from the neural retina and pigmented epithelium showed that cells from *beg* embryos started transdifferentiation about a week earlier, had a higher rate of transdifferentiation, and also retained their transdifferentiation potential significantly longer than cells from normal embryos.

Blind, cataracts (*bc*). This mutation was discovered in a research strain carrying a pericentric chromosomal inversion (Bitgood et al., 1986). Data obtained indicated that the mutation was autosomal recessive and not associated with the inversion. Affected chicks were blind at hatch and showed evidence of posterior subcapsular lenticular opacities or cataracts. The cataracts progressed from faint opacities to more visible opacities as the chick grew. No further information has been published, but the mutation has been maintained (Bitgood, 1988, personal communication).

Hereditary blindness (*bli*). This autosomal recessive mutation found in White Leghorns (Hutt, 1935) is the earliest reported ocular mutation in chickens. Affected chicks were blind at hatch and showed no pupillary reflex. The chicks also had unilateral or bilateral bulging of the eyes, but the bulging was asymmetrical even if it was bilateral. The right and left eyes were usually different in size and accompanied by unilateral atrophy of the optic nerve. Blind males produced normal semen and blind females had normal egg production. This mutation has not been maintained.

Partial retinal dysplasia and degeneration (*rdd*). This blindness trait was first observed in Scotland in chicks from a commercial cross of Barred Plymouth Rocks and Rhode Island Reds (Siddens, 1981, personal communication). Both parental stocks were imported from the U.S.A. via South Africa. No blindness had been observed in the parental stocks prior to importation, but subsequently blind chicks (up to 30 percent in a hatch) were observed in the Rhode Island Red pure line. Despite selection against the blind trait, the frequency of blind chicks remained at about four percent in the commercial cross and the Rhode Island Red pure line in subsequent generations.

The trait was first described by Randall and McLachlan (1979) as involving retinal degeneration, and further characterized by Wilson et al. (1982) who reported that the condition behaved like a single recessive mutant. The gene symbol *rdd* was proposed for partial retinal dysplasia and subsequent degeneration. Randall et al. (1983) outcrossed the blind hens from the commercial stock to Brown Leghorns and carried out testmatings to confirm that the mutation was autosomal recessive.

At hatching the affected chicks had limited vision but were less active than normal sighted chicks. By six months of age the majority of affected chicks made no response to visual stimuli. Electroretinograms performed on week-old *rdd* chicks indicated that vision was poor but not yet lost, and the sensitivity of rods and cones was greatly diminished.

Such results were further confirmed by standard visual acuity tests. Observations of histological sections from embryos and hatched chicks revealed abnormalities in the retina extending from the pigmented epithelium (RPE) to the inner nuclear layer. Starting on the eighth day of incubation, holes were observed in the RPE. These holes grew in number and size as incubation progressed, but unlike the retinal holes of *beg* chicks, these RPE holes disappeared within a week after hatching. In hatched chicks a progressive degeneration of photoreceptor cells was observed. Kondoh et al. (1980) examined *in vitro* cultures of cells of *rdd* chick retinas and observed a higher rate of programmed cell death than that from normal chick embryos. They have also reported that neural retinal cells obtained from a 7-day *rdd* embryo had a 30 percent slower growth rate compared to the cells from normal age-matched embryos.

The *rdd* chickens may be useful as a model to study ocular abnormalities such as retinitis pigmentosa in humans, but further characterization of the *rdd* retina seems necessary (Randall et al., 1983).

Blindness, rods and cones (*rc*). Blind chicks were first observed in a line of chickens carrying an ethyl methanesulfonate (EMS) induced translocation involving the Z chromosome and chromosome 3 (Cheng et al., 1978). Through testmatings it was determined that the blindness was caused by a single autosomal recessive mutation which was later named rods and cones with gene symbol *rc* (Cheng et al., 1980). The *rc* chickens were blind at hatch. Although they hatched normally, they did not respond to visual cues and exhibited random head bobbing and twisting to balance themselves. Mutant chicks had no measurable retinal signals for either the light-adapted or the dark-adapted ERGs on the day of hatching and at any time posthatching (Ulshafer et al., 1984). Heterozygous chickens appeared to have normal vision, but ERGs of nine heterozygous chickens measured at ten months of age were outside the range of variation for normal chickens (Cheng & Pang, 1988). While it was likely that the mutation was induced by EMS, there was no conclusive evidence. Linkage studies showed that the mutation was not associated with the break-attachment point of the translocation. A line of chickens carrying the *rc* gene, but not the translocation, was established and maintained for research purposes.

Cheng et al. (1980) reported that there was agenesis of rods and cones in the retina of mutant chick embryos. Further light microscopy study of newly hatched and older mutant chicks (Wolf et al., 1981) indicated that rods and cones were present in the newly hatched chicks, but that the photoreceptor components were significantly abnormal at hatching. Later, Wolf (1982) examined mutant embryos that were older than 13 days of incubation and reported that no obvious differences were visible by light microscopy of the mutant embryonic eyes compared to the normal. However, he also reported that newly hatched chicks showed lack of development of the complete complement of crystalloid bodies in the inner segments and loss of organization of the components in the photoreceptor space. Progressive degeneration of the outer and inner segments started on the first day after hatching. More recent histological and ultrastructural studies of the *rc* retina by Ulshafer et al. (1984), and Ulshafer and Allen (1985a) further changed the picture. They reported that the retinal morphology of newly hatched mutant chicks was comparable to that of normal chicks of the same age, and no pathological changes were evident. Although there were signs of disorganization during the first week posthatch, significant degeneration of the retina only started during the second week after hatching. Because of the differences between their findings and those reported in the literature, Ulshafer et al. (1984) referred to the mutant as *rd*.

While these reports seem to show conflicting results, they may indicate that there are individual variations in the phenotypic expression of the *rc* gene (only a small number of chicks or embryos were examined in each study), and/or that the expression of the *rc* gene has been modified by natural selection in subsequent generations after the initial discovery. Mutant individuals which carried modifying genes that would delay or moderate the expression of the *rc* gene would survive better than mutants not carrying these modifying genes. As a result, mutant phenotype may change over generations. Despite these variations, no mutant chicks examined showed ERG responses at hatch.

Regardless of when the degeneration process started, by 6-8 months after hatching, only a few sporadic photoreceptors remained. Photoreceptors degenerated along a central to peripheral gradient (Ulshafer et al., 1984). Most of the remaining photoreceptors were double cones. In the outer nuclear layer the majority of the degenerated cells had been replaced by processes of the Mueller cells. At this stage changes were also noted in the RPE (Ulshafer and Allen, 1985b). Cheng (unpublished) examined retinas of two 14-week old heterozygotes using light microscopy and found distended photoreceptors in both, although there were no signs of degeneration. Intensive studies had been carried out to determine the etiology and pathogenesis of the retinal degeneration of the *rc* chicken (Ulshafer et al., 1985; Kelley et al., 1985; Ulshafer et al., 1986; Spoerri et al., 1986; Lee et al., 1987; Allen & Ulshafer, 1987; Semple-Rowland et al., 1988; Lee et al., 1988).

Pang et al. (1988) measured the concentrations of melatonin in the retina, serum, and pineal gland of mutant chicks at various ages from 4-14 weeks posthatching, and used age-matched sighted heterozygous chicks as controls. Both the mutants and the controls showed diurnal variation of retinal melatonin levels at all ages, with high levels at night. Mutant chicks had significantly lowered (38-75 percent) retinal melatonin levels in the dark when compared to controls at the same age. The decrease in retinal melatonin in the mutant chicks seemed to correlate well with the progressive degeneration of photoreceptors in their retinas. There were also diurnal rhythms of pineal melatonin with high levels in the dark period for both the mutants and the controls. Furthermore there was no significant difference in the levels of pineal melatonin between the two groups. The results indicated that synthesis and storage activities of melatonin in the pineal of the two groups were similar and the mutants did not show any pathological abnormalities. However, when serum melatonin of these same birds was examined, mutant chicks had significantly lower circulating melatonin levels at mid-dark than those of the controls at the same age. Apparently, although the *rc* gene has not affected the synthesis and storage activities of the pineal, it may have pleiotropically affected the secretory activities of the gland (Cheng and Pang, 1988).

The *rc* gene affects the photoreceptors and retinal melatonin of the chicken and also has pleiotropic effects on circulating melatonin. The line of chickens carrying this mutation should be useful in research concerning the synthesis, regulation, and functions of melatonin both as a neuromodulator (ocular) and as a hormone (systemic), as well as research on the etiology and pathogenesis of retinal degeneration. Should the heterozygotes turn out to have an intermediate phenotype in some of the traits affected by this gene, and the dosage effect can be measured, then *rc* chickens may be an even more valuable model.

Ocular amelanosis and retinal degeneration (DAM line). DAM stands for delayed amelanosis. The line of chickens with this condition originated from a hen of the experimental Brown line at the University of Massachusetts in 1971 (Smyth et al., 1977). This particular hen had normally pigmented juvenile feathering, but her adult plumage was completely white and subsequently the hen became blind. The DAM line was developed by simultaneous outcrossing and *inter se* matings of related individuals of the Brown line, and the subsequent combining of these two sublines after three generations (Smyth et al., 1981). The incidence of delayed amelanosis was 60 percent with 25.6 percent of the abnormal birds being blind. By selecting blind individuals for matings in the next three generations, the frequency of blindness was increased to 81 percent and the BDAM (blind, delayed amelanosis) subline was established. Genetic segregation indicated that blindness was closely associated with delayed amelanosis, both traits seemed to be autosomal, and neither segregated in a simple Mendelian manner (Boissy et al., 1977). The outcross matings showed the polygenic nature of these traits but the number of genes involved may be small. The outcross matings also indicated that the traits may be related to the *E* locus.

Further studies into the etiology of these pathological conditions revealed that the DAM chickens had a hyperactive immune system, particularly in B-cell functions (Lamont and Smyth, 1981; Lamont and Smyth, 1984), and had morphologically abnormal melanocytes (Boissy et al., 1984). Postnatally the melanoblasts of the DAM chickens differentiated with each molt into progressively more abnormal melanocytes. These abnormal melanocytes were eliminated from regenerating feathers and the choroid of the eye in an autoimmune fashion resulting in loss of melanin pigments from these tissues and a massive infiltration of mononuclear leukocytes into the supporting connective tissues (Boissy et al., 1984). The ocular amelanosis affected the RPE and led to retinal degeneration and blindness (Fite et al., 1982). The DAM chickens appear to be a valuable model for vitiligo and melanin-related eye diseases. Further discussion of the DAM chickens can be found in Chapter 5 and under autoimmune thyroiditis in this chapter.

Blindness, hyperplasia of lens epithelium (Hy-1, Hy-2 lines). 'Hy' is an abbreviation of hyperplastic. Hy-1 and Hy-2 are two unrelated lines of chickens, both selected for fast growth rate. Hy-1 was originally derived mainly from Cornish Games but had been under stringent selection for growth rate for more than 20 years. Hy-2 is a cross between two lines originating mainly from White Plymouth Rocks; the parental stocks had similar selection history to the Hy-1 line (Clayton, 1975). Birds in these two lines had abnormal lens growth and ring cataracts that led to blindness. The normal lens of chickens has an anterior epithelial layer, with mitosis confined to a germinal zone just anterior to the equator of the lens. In Hy-1 and Hy-2 chicks there was an excessive growth of epithelial cells from the germinal zone forming multiple-layered and buckled lens epithelia. Although the outermost epithelial layer appeared to be normal compared to the epithelial layer of normal chicks, some of the cells of the excessive inner layers differentiated into short fibre-like cells, which elongated perpendicular to the plane of the epithelial sheet, and were not oriented with respect to the body of the lens (Clayton, 1975; Odeigah et al., 1979). It was hypothesized that the stringent selection for fast growth rate in the two lines had altered the cell surface and cell growth properties, and the control of cytodifferentiation in the lens as well as in other body tissues (Pritchard and Clayton, 1978). The Hy-1 and Hy-2 chickens have been useful in research concerning cell growth, cell membrane functions, and cytodifferentiation (Clayton et al., 1976a; Clayton et al., 1976b; Eguchi et al., 1975; Truman et al., 1976).

Coloboma of the iris. Coloboma is a form of defective formation of the eye, characterized by an indentation of the iris. The defect may be the result of inadequate blood supply to the eye during embryonic development (Stroeva, 1961). Birds with coloboma appeared to be normal in all other respects. Wilcox (1958) attempted to study the mode of inheritance of coloboma in chickens by mating a male with coloboma to four females with coloboma, four normal females whose dam had coloboma, and four normal females whose full-sib had coloboma. From the progeny of these matings, two males and five females with coloboma were mated and the second generation progeny were also screened for coloboma. The results indicated that when both parents had coloboma (both in the first and in the second generation), the frequency of coloboma in the progeny was 27 percent. When only one of the parents had coloboma, the frequency in the progeny was about 20 percent. Males had a significantly lower incidence of coloboma than did females. These genetic tests were not rigorous enough to evaluate the mode of inheritance of coloboma except to indicate that the trait was not inherited in a simple Mendelian manner.

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Chapter 11. LETHAL MUTANT TRAITS IN CHICKENS

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Over 100 chicken mutant loci have been shown to have lethal effects. Lethal action may occur during the incubation period or at various times during later life. Lethality may be inevitable (*obligate*) or dependent upon environmental factors (*facultative*). The standard reference sources concerning embryonic lethals are Landauer's (1967) monograph *The Hatchability of Chicken Eggs as Influenced by Environment and Heredity* and its supplement (1973), and Hadorn's (1961) excellent book *Developmental Genetics and Lethal Factors*. An excellent review paper by Cole (1969) deals with nonembryonic lethals.

Lethal traits can be grouped by inheritance mode and degree of lethality as follows:

1. Dominant gene with obligate recessive lethal action. The homozygotes are too abnormal to develop and hatch, but the heterozygotes, although abnormal, are viable. Matings segregating for this type of trait give a characteristic 2:1 ratio. There are several of this type of lethal in the chicken. These have been described in other chapters and are listed in Table 11.1 for reference.
2. Obligate semilethal trait. Traits of this type may be either dominant or recessive, and although their time of lethal action is usually during embryonic life, it also may occur later in life. Lethals of this type allow a varying proportion of affected individuals to survive. The proportion surviving may depend upon environment and/or modifying genes. When lethality is between 50 and 100 percent this type of trait is referred to as semilethal, and when lethality is less than 50 percent it is called subvital. There are a number of chicken traits that fit into this category. They have been described in other chapters and are listed here in Table 11.1.
3. Facultative lethal trait. Traits that fall into this category are generally not lethal due to direct action of the mutant gene. The characteristics of the trait, however, are such that the individuals do not survive well in what would be considered a normal environment. Thus, special effort must be made to provide an extremely favorable environment to keep mortality at a minimum. There are many chicken traits that are facultative lethals. They have been described in other chapters and are listed in Table 11.1.
4. Posthatch obligate lethal. The time of action of this type of obligate lethal is after the chick has hatched, and it may even be as late as adulthood. These lethals have also been described in other chapters and are listed here in Table 11.1.
5. Autosomal recessive obligate embryonic lethal. This type of lethal has no effect in the heterozygote but it is lethal to the homozygote in either early or more often late embryonic life. Early embryonic lethality may be recognized because it is associated with or linked to some identifiable trait such as color or morphology. In later embryonic life, the lethal trait is usually recognized by gross abnormalities. In both cases, hatchability is reduced by about 25 percent. Mutants of this type, of which there are 35, will be described in this chapter, with the exception of the trait riboflavinuria (*rd*) which is discussed in Chapter 32.
6. Sex-linked obligate embryonic lethal. This type of lethal affects female embryos only. Carrier males are unaffected by it. It is usually detected by a 50 percent deficiency of hatched females. If its action is late in embryonic development, then abnormalities will generally be evident. Seven mutants of this type are described in this chapter.

Table 11.1. Lethal mutant traits described in other chapters.

Trait	Gene symbol	Chapter	Trait	Gene symbol	Chapter
Dominant traits with obligate recessive lethal action					
White wing	*Ww, ww*$^+$	5	Creeper	*Cp, cp*$^+$	7
Ear-tufts	*Et, et*$^+$	6	Cornish lethal	*Cl, cl*$^+$	7
Flightless	*Fl, fl*$^+$	6	Ametapodia	*Mp, mp*$^+$	7
Obligate semilethal traits					
Albinism	*a, A*$^+$	5	Rumplessness	*Rp, rp*$^+$	7
Imperfect albinism	*s*^{al}, *s*$^+$	5	Recessive rumplessness	*rp-2, Rp-2*$^+$	7
Extended black	*E, e*$^+$	5	Recessive polydactyly	*po-2, Po-2*$^+$	7
Woolly	*wo, Wo*$^+$	6	Multiple trait semilethal	*psp, Psp*$^+$	7
Sex-linked naked	*n, N*$^+$	6	Shankless	*shl, Shl*$^+$	7
Ichthyosis	*dehy, Dehy*$^+$	6	Micromelia-Hays	*mm-H, Mm-H*$^+$	7
Scleroderma	*sd, Sd*$^+$	6	Sex-linked wingless	*wl, Wl*$^+$	7
Ear-tufts	*Et, et*$^+$	6	Cleft palate	*polygenic*	7
Naked neck	*Na, na*$^+$	6	Supernumerary ribs	*polygenic*	7
Apterylosis	*Ap, ap*$^+$	6	Sex-linked nervous disorder	*sln, Sln*$^+$	9
Ottawa naked	*nk, Nk*$^+$	6	Tipsy	*tip, Tip*$^+$	9
Edema	*ed-1, Ed-1*$^+$; *ed-2, Ed-2*$^+$	6	Microphthalmia	*mi, Mi*$^+$	10
Short mandible	*sm, Sm*$^+$	7	Dominant microphthalmia	*Mi-2, mi-2*$^+$	10
Short upper beak	*su, Su*$^+$	7	Microphthalmia-3	*mi-3, Mi-3*$^+$	10
			Microphthalmia-4	*mi-4, Mi-4*$^+$	10
Facultative lethal traits					
Apterylosis	*Ap, ap*$^+$	6	Exencephaly	*polygenic*	7
Sex-linked naked	*n, N*$^+$	6	Crippling anomaly	*polygenic*	7
Flightlessness	*Fl, fl*$^+$	6	Ventricular septal defect	*polygenic*	7
Sleepy-eye	*se, Se*$^+$	6	Shaker	*sh, Sh*$^+$	9
Scaleless	*sc, Sc*$^+$	6	Shaker-2	*sh-2, Sh-2*$^+$	9
Ottawa naked	*nk, Nk*$^+$	6	Tipsy	*tip, Tip*$^+$	9
Wiry	*wi, Wi*$^+$	6	Arched neck	*polygenic*	9
Woolly	*wo, Wo*$^+$	6	Recessive dwarfism	*td, Td*$^+$	10
Porcupine	*pc, Pc*$^+$	6	Gasper	*ga, Ga*$^+$	10
Dactylolysis	*dac, Dac*$^+$	6	Hereditary blindness	*bli, Bli*$^+$	10
Short upper beak	*su, Su*$^+$	7	Microphthalmia	*mi, Mi*$^+$	10
Short mandible	*sm, Sm*$^+$	7	Dominant microphthalmia	*Mi-2, mi-2*$^+$	10
Crooked neck	*crn, Crn*$^+$	7	Microphthalmia-3	*mi-3, mi-3*$^+$	10
Shankless	*shl, Shl*$^+$	7	Microphthalmia-4	*mi-4, mi-4*$^+$	10
Ametapodia	*Mp, mp*$^+$	7	Blindness, enlarged globe	*beg, Beg*$^+$	10
Congenital leg anomaly	*cla, Cla*$^+$	7	Blind, cataracts	*bc, Bc*$^+$	10
Congenital crippling	*coc, Coc*$^+$	7	Retina dysplasia and degeneration	*rdd, Rdd*$^+$	10
Congenital perosis	*cop, Cop*$^+$	7	Blindness, rods, and cones	*rc, Rc*$^+$	10
Posthatch obligate lethal traits					
Blistered foot lethal	*bf, Bf*$^+$	6	Congenital loco	*lo, Lo*$^+$	9
Stringy	*st, St*$^+$	6	Sex-linked lethal	*xl, Xl*$^+$	9
Sparrow head	*sph, Sph*$^+$	7	Jittery	*j, J*$^+$	9
Micromelia-Hays	*mm-H, Mm-H*$^+$	7	Nervous disorder	*hnd, Hnd*$^+$	9
Crazy	*cy, Cy*$^+$	9	Paroxysm	*px, Px*$^+$	9
Faded shaker	*fs, Fs*$^+$	9	Congenital tremor	*polygenic*	9
			Atresia isthmi	*Aro, aro*$^+$	10

Some traits might easily fit under several of these classifications. For example both the ear-tuft and ametapodia mutants are obligate lethals with the homozygous dominants dying during embryonic development. However the ear-tuft gene is semilethal to 20-42 percent of the heterozygous ear-tufted embryos. In the case of the heterozygous ametapodial chicks, the gene is a facultative lethal because all would die unless extreme management practices were observed.

In this chapter the autosomal recessive and sex-linked obligate embryonic lethals will be reviewed. They will be covered under six headings. There are five *early embryonic lethals*, two of them autosomal and three sex-linked. Ten of the traits are grouped as *polydactylous embryonic lethals*, all of which have abnormal digit patterns. An additional five are *hypodactylous embryonic lethals* with greatly reduced limbs. There are seven traits that are *chondrodystrophic embryonic lethals*. These display a shortening of the long bones. Eight traits are *facial embryonic lethals* affecting mainly the beak. Six more are *miscellaneous embryonic lethals* covering an array of characterizations.

EARLY EMBRYONIC LETHALS

The five lethal traits whose action occurs during the first few days of embryo development are listed in Table 11.2.

Recessive white lethal (*l*). The recessive white lethal was the first of over 40 embryonic lethals to have been reported in the domestic fowl. Dunn (1923) found that when the offspring of a White Wyandotte x Pit Game cross were used in backcrosses approximately half of the white progeny were missing. The resulting 2:1 ratios were consistent with the hypothesis that an autosomal recessive lethal gene was closely linked to the recessive white locus. This hypothesis was further supported by the observations that there was extremely high early embryonic mortality in the pure Wyandotte stock used in the original cross, in outcrosses there was high embryo viability, low viability returned when backcrosses were made to the Wyandotte stock, and mortality was very high among chicks hatching from crosses involving the lethal. This also suggested that heterozygotes for the lethal gene were less viable. Analysis of color segregation ratios among embryos suggested that death occurred early in incubation, sometime prior to 16 days. Warren (1933) reported a similar condition in White Wyandottes. He further made the point that poor hatching quality was characteristic of White Wyandottes. Dunn (1923) and Warren (1933) were

Table 11.2. Early embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Recessive white lethal	*l*, L^+	Dunn (1923)
Blood ring	*blr*, Blr^+	Savage et al. (1988)
Sex-linked recessive		
Early sex-linked lethal-Bernier	*sex*, Sex^+	Bernier and Arscott (1972)
Prenatal lethal	*pn*, Pn^+	Somes and Smyth (1967)
Ladykiller	*lk*, Lk^+	Sheridan (1964)

probably both working with the same locus, to which Dunn (1923) assigned the gene symbol *l*. It is also possible that this lethal may have been an allele at the recessive white locus rather than a closely linked independent locus.

Blood ring (*blr*). Savage et al. (1988) have reported another autosomal recessive gene that causes lethality very early in incubation. They named it blood ring and assigned the gene symbol *blr*. This gene was found in White Leghorn stock. At 42 h of development mutant embryos appeared normal, but by 48 h their development had slowed down (stage 12 vs. 13). At 72 h cessation of development was usually complete. Stage 17 embryos were surrounded by a blood ring with uncoalesced blood islands, and the sinus terminalis was engorged with erythrocytes. This recessive gene was detected in three commercial flocks with gene frequencies ranging from 0.08 to 0.16. It was suggested that a defective gene product associated with cardiac morphogenesis and vitamin A utilization might have been involved.

Early sex-linked lethal-Bernier (*sex*). Three sex-linked conditions have been reported which have their lethal effects early in the incubation period. The first of these was briefly mentioned by Bernier and Arscott (1972). It occurred in their selected sex-linked dwarf line. A male mated to 50 females sired 437 chicks of which 297 were males and 140 were females. This sex ratio was not significantly different from the 2:1 ratio that would be expected if a sex-linked recessive lethal gene were operative in this mating. It was reported that the missing female zygotes apparently died very early in incubation. The gene symbol *sex* has been assigned this condition (Somes, 1978); however, it is also possible that this lethal condition may be the same as one or both of those whose descriptions follow.

Prenatal lethal (*pn*). Somes and Smyth (1967) found an early-acting sex-linked lethal in a Buff Brahma bantam stock. Their extensive data (Somes and Smyth, 1967; Somes, 1969) showed that the time of gene action was during embryonic stages 18-20, which are found at 3-3.5 days of incubation. Linkage studies showed close association with both the *K* and *S* loci. They assigned the name prenatal to this lethal, along with the gene symbol *pn*.

Ladykiller (*lk*). Another early sex-linked lethal very similar to the prenatal lethal was reported by Sheridan (1964, 1979). It was isolated from a broiler flock. Its major time of action was between stages 18 and 21, and it was located at the same end of the sex chromosome as the prenatal lethal. However, Sheridan (1979) reported that the ladykiller gene, to which he assigned the gene symbol *lk*, was located on the opposite side of the feathering locus (*K*) from that of both the dwarf (*dw*) and prenatal (*pn*) loci, and thus presumably it was at a separate locus from that of the prenatal lethal.

POLYDACTYLOUS EMBRYONIC LETHALS

The ten polydactylous embryonic lethals are listed in Table 11.3. Nine of these are autosomal and one is sex-linked; all of them are recessive.

Diplopodia (*dp-1, dp-2, dp-3, dp-4, dp-5*). A lethal form of polydactylism, first reported by Taylor and Gunns (1947), has since been followed by four other reports of similar polydactylous lethals (Landauer, 1956b; Taylor, 1972; Abbott and Kieny, 1961;

Table 11.3. Polydactylous embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Diplopodia-1	*dp-1*, *Dp-1*$^+$	Taylor and Gunns (1947)
Diplopodia-2	*dp-2*, *Dp-2*$^+$	Landauer (1956b)
Diplopodia-3	*dp-3*, *Dp-3*$^+$	Taylor (1974)
Diplopodia-5	*dp-5*, *Dp-5*$^+$	Olympio et al. (1983)
Splitfoot	*sf*, *Sf*$^+$	Abbott and Goetinck (1960)
Eudiplopodia	*eu*, *Eu*$^+$	Rosenblatt et al. (1959)
Talpid-1	*ta-1*, *Ta-1*$^+$	Cole (1942)
Talpid-2	*ta-2*, *Ta-2*$^+$	Abbott et al. (1960)
Talpid-3	*ta-3*, *Ta-3*$^+$	Hunton (1960)
Sex-linked recessive		
Diplopodia-4	*dp-4*, *Dp-4*$^+$	Abbott and Kieny (1961)

Olympio et al., 1983). In order to differentiate this form of polydactyly from other forms in the domestic fowl, and because supernumerary metatarsals are a feature of it, Taylor and Gunns (1947) adopted the term diplopodia for this particular condition which caused doubling of the structure of the foot and was lethal.

Although there is some variability within and between these five types of diplopodia, their main effects are quite similar. In general, toes number from five to eight per foot with six per foot being the most common. One complement of three or four toes is in the position of normal digits II, III, and IV, while the second complement is on the medial side of the tarsometatarsus anywhere along its length. The hallux (digit I) is absent as are its related metatarsal bones. Supernumerary metatarsal bones are also a feature although there is extreme variability in their numbers, ranging from one to four. When asymmetry of toe numbers is present, the right leg generally possesses the greater number of toes. Taylor (1972) placed four of the diplopods in rank of severity as follows: *dp-3* < *dp-1* < *dp-4* < *dp-2*. The description of diplopodia-5 (Olympio et al., 1983) would seem to place it in a severity range similar to that of diplopodia-3 (Figure 11.1). Other phenotypic influences of these diplopod genes are supernumerary wing digits, shortened long bones and upper beak, reduced body weight and length, and an inability to hatch. Most diplopod embryos die during the last days of incubation, with only a few pipping. Diplopodia-2 embryos had a greater percentage die before 18 days of incubation than did the other four types. Only rarely did any diplopod chicks hatch, and when they did, they died within a few days. However, Taylor (1958) did manage to raise one female and two males to maturity. One male was fertile, and when mated by A.I. to carrier females it was shown to be homozygous for diplopodia-1. Kieny and Abbott (1962) also studied the relative growth of the fibula and tibiotarsus in diplopodia-4.

The original diplopod lethal was shown to be inherited as an autosomal recessive (Taylor and Gunns, 1947), as was the second one studied by Landauer (1956b). Landauer (1956b) also tested these two types of diplopods for allelism and found them to be completely independent. Thus, he assigned the symbol *dp-1* to the Taylor-Gunns diplopod and *dp-2* to his own. Taylor's (1974) diplopodia-3 mutant, also an autosomal recessive, was tested against *dp-1* and found to segregate independently. As diplopodia-3 had been previously assumed to be independent, it had already been assigned the gene symbol *dp-3*. Diplopodia-4 proved to be inherited as a sex-linked recessive, and it was assigned the symbol *dp-4* (Abbott and Kieny, 1961). Lastly, diplopodia-5 proved to be an autosomal recessive (Olympio et al., 1983). It was tested against *dp-3* and shown to be nonallelic, receiving the gene symbol *dp-5*.

During the original studies of diplopodia-1, two atypical carrier females were discovered that produced only 13.6 percent diplopods (Taylor and Gunns, 1947). These two birds were further studied, and three selection lines were developed which differed in the percentage of diplopods that were produced from carrier x carrier matings; line I produced 20.4 percent, line II 12.7 percent, and line III 6.1 percent diplopods (Taylor et al., 1959). These lines were used to study the effect that residual genotype exerted over the control of penetrance and expressivity of a single gene trait (Abbott, 1959a, 1959b).

Splitfoot (*sf*). A phenotypic expression, which mainly appeared as malformation of the feet, was discovered by Abbott and Goetinck (1960) in a White Leghorn family and was named splitfoot (Figure 11.1). The hallux (digit I) and digit II were generally altered, with digits III and IV remaining unaltered. The effects on the foot were varied and could be placed in five classes as follows: only the hallux was affected; digit II was split in two, each digit having three phalanges; digit II was split, but the lateral bifurcation had three and the median had four phalanges; both parts of digit II had four phalanges; and the foot possessed five digits with the hallux being absent. Foot arrangements were not necessarily bilateral. Other abnormalities associated with the splitfoot phenotype were reduced premaxilla, incomplete eyelids, micromelia, unutilized albumen at time of death, and embryonic death between 17-19 days of incubation. Sometimes the second tarsometatarsal bone was duplicated.

The splitfoot phenotype was found in 30.2 percent of 291 embryos and chicks from carrier matings. Although this total ratio deviated significantly from 3:1, 12 of the 13 individual matings yielded normal ratios and thus it was assumed that a single autosomal recessive gene was involved. The gene symbol *sf* was suggested.

Eudiplopodia (*eu*). A lethal mutation which is unique among the polydactylous mutants was described by Rosenblatt et al. (1959). It had occurred in a White Leghorn flock. This condition, which has been named eudiplopodia, consists in its most common expression of two complete sets of digits, with one set above the other. The number of toes per abnormal foot varies from five to nine. No other polydactylous mutants have their supernumerary digits located dorsally to the normal ones. Weaker expressions of eudiplopodia consist of replication of only one or two toes. Toenails on the extra digits are cone-shaped, lacking the concave ventral surface of normal toenails. This condition basically affects the legs, because only three percent of eudiplopods had any wing bone manifestations; however, a large proportion of eudiplopod embryos exhibited twinning or more extensive duplication of primary flight feathers in one or more follicles (Fraser and Abbott, 1971a). This condition is always lethal during the terminal stages of incubation.

Eudiplopod embryos were found to develop secondary apical ectodermal ridges during stages 22 or 23 (Goetinck, 1964; Fraser and Abbott, 1971b). These lead to mesodermal outgrowths and the formation of supernumerary digits. Reciprocal transplants localized the site of defect to the ectoderm. It was further demonstrated that the number of extra toes was dependent on level of incubation temperature (Fraser and Abbott, 1971a). Low temperatures suppressed the number of toes, while high temperatures increased their numbers.

Genetic studies (Rosenblatt et al., 1959) showed that when carrier birds were mated *inter se* 454 normal and 148 eudiplopod embryos were produced, a close fit for the expected 3:1 ratio for an autosomal recessive. The gene symbol *eu* was assigned. The eudiplopod gene was also tested for linkage with the diplopodia-1 trait and found to be nonallelic to it (Rosenblatt et al., 1959).

Talpid (*ta-1, ta-2, ta-3*). A lethal condition known as talpid, named after the Latin world *talpa* because the feet and wings of embryos with this condition resemble those of a mole, has been reported three separate times. Cole (1942) described the first of these, talpid-1, from White Leghorn stock. Later, Abbott et al. (1960) reported on talpid-2 also from White Leghorns, and Hunton (1960) found talpid-3 in a Light Sussex flock. The genetic basis of each of these talpids has been shown to be that of a single autosomal recessive. Unfortunately, none of these three mutations has been tested against any of the others, thus it is not known whether they represent the same or different mutated loci. Talpid-2 has some features which make it somewhat different from the other two. At the present time, the gene symbols *ta-1*, *ta-2* and *ta-3* are used to indicate these three lethals.

Morphologically all three talpids are quite similar. The length of leg and wing proximal bones is greatly reduced, and there is duplication of the distal digit bones. Each foot and wing has syndactylous digits forming a cupped and webbed or hand-like structure. The vertebral column is shortened and development of the face is retarded. Extreme visceral ectopia is prominent in older embryos and subcutaneous edema appears as large blebs over the thigh region, neck, and head. Inman (1946) made histological studies of talpid-1 and found pituitary development abnormalities. Similar histologic defects of the head were described for talpid-3 (Ede and Kelly, 1964a) but could not be shown for talpid-2 (Abbott et al., 1960). The time of lethal action was reported as 8-10 days for talpid-1 (Cole, 1942), 10-14 days for talpid-2 (Abbott et al., 1960), and about 14 days or earlier for talpid-3 (Ede and Kelly, 1964a). The complex pattern of developmental abnormalities has further been studied for limb morphogenesis in talpid-2 (Goetinck and Abbott, 1964; Niederman and Armstrong, 1972), and for head region (Ede and Kelly, 1964a), trunk and limbs (Ede and Kelly, 1964b; Hinchliffe and Ede, 1967), cell adhesion and movement (Ede and Agerbak, 1968), bone and cartilage (Hinchliffe and Ede, 1968), feather morphogenesis (Ede et al., 1971), and patterns of cell division and cell death (Ede and Flint, 1972) in talpid-3 embryos.

The characteristic talpid shovel-like appendages seem to result from abnormal cell adhesion and movement and a reduction of normal cell death. Hinchliffe and Ede (1967) showed that the mesenchymal cells of talpid embryos are protected against the normal necrosis and degeneration that is characteristic of digit development. Cell motility studies of talpid-3 mesenchyme cells (Ede and Agerbak, 1968) lead to the conclusion that mutant talpid-3 cells have abnormal adhesive characteristics. It was suggested that this defect could lead to the distorted skeletal condensations characteristic of talpid feet and wings. A computer simulation of limb bud growth verified the resulting limb shape (Ede and Law, 1969). However, Niederman and Armstrong (1972), using the talpid-2 mutant, concluded that the mesodermal cells of talpid-2 do not differ from normal mesoderm cells in their adhesiveness.

HYPODACTYLOUS EMBRYONIC LETHALS

The five hypodactylous embryonic lethals are listed in Table 11.4. Four are autosomal and one is sex-linked. All are recessive.

Wingless (*wg*). Waters and Bywaters (1943) described a mutant that occurred in White Leghorn embryos. Because of its extreme effect on the wings, they termed it wingless.

Table 11.4. Hypodactylous embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Wingless	*wg*, *Wg*$^+$	Waters and Bywaters (1943)
Wingless-2	*wg-2*, *Wg-2*$^+$	Zwilling (1956)
Limbless	*ll*, *Ll*$^+$	Prahlad et al. (1979)
Stumpy	*stu*, *Stu*$^+$	Abbott et al. (1966)
Sex-linked recessive		
Coloboma	*cm*, *Cm*$^+$	Abbott et al. (1970)

Most defective embryos showed no wing development at all, although occasional vestigial wing appendages appeared on one or both sides (Figure 11.1). This absence of wing buds could be detected as early as four days of incubation. Abnormalities of legs and toes included missing or duplicated toes, syndactylous third and fourth toes, misplacement of the first toe, and rudimentary scales. In most cases there was no development of the metanephros or definitive kidney, and only a small part of the lung tissue was present. All wingless embryos also had clubbed down. Studies of early embryos (Zwilling, 1949) showed that absence of wings was due to a failure in the development of the apical ectodermal ridge of the wing bud. A similar failure occurring later in development in the hindlimb buds led to the less dramatic defects of the legs and toes. Many of the wingless embryos survived to hatching time but none ever hatched. Lack of kidney development was due to failure of the ureteric buds to develop, which in turn prohibited metanephric kidney differentiation.

This wingless syndrome was shown to be inherited as a single autosomal recessive trait when 30 wingless embryos were found among 141 progeny of heterozygous carrier parents. Chicks heterozygous for this gene appeared to be perfectly normal. Hutt (1949) proposed the gene symbol *wg* as being appropriate for this trait.

Wingless-2 (*wg-2*). A second and quite similar wingless mutant was reported by Zwilling (1956), with its biggest difference being the presence of lungs, more frequent leg involvement, and a more consistent completeness of winglessness. Because of the loss of the *wg* gene, it could not be ascertained whether the new wingless was a repeat mutant or was independent of the original Waters-Bywaters mutant. The Zwilling wingless is now known as wingless-2 and has the gene symbol *wg-2* (Somes, 1980).

Wingless-2 embryos were also unable to maintain a wingbud apical ectodermal ridge or to develop ureteric buds. Results from interchange of limb components (Zwilling, 1956) and tissue composite grafts (Zwilling, 1974) seemed to indicate that limb development depends on a reciprocal interaction between the mesoderm and the apical ectoderm, and that with the wingless mutant the mesoderm factor, a maintenance factor for the apical ectoderm, was deficient. Loewenthal (1957) reported data that indicated the existence of degenerative events in the apical ectoderm and underlying mesoderm, while Rubin, MacCabe, and MacCabe as quoted by Saunders (1972) described the absence of the 'zone of polarizing activity' in wingless embryos. Such a defect could account for the lack of wing development in wingless embryos.

Limbless (*ll*). This lethal mutant, reported by Prahlad et al. (1979) in a Rhode Island

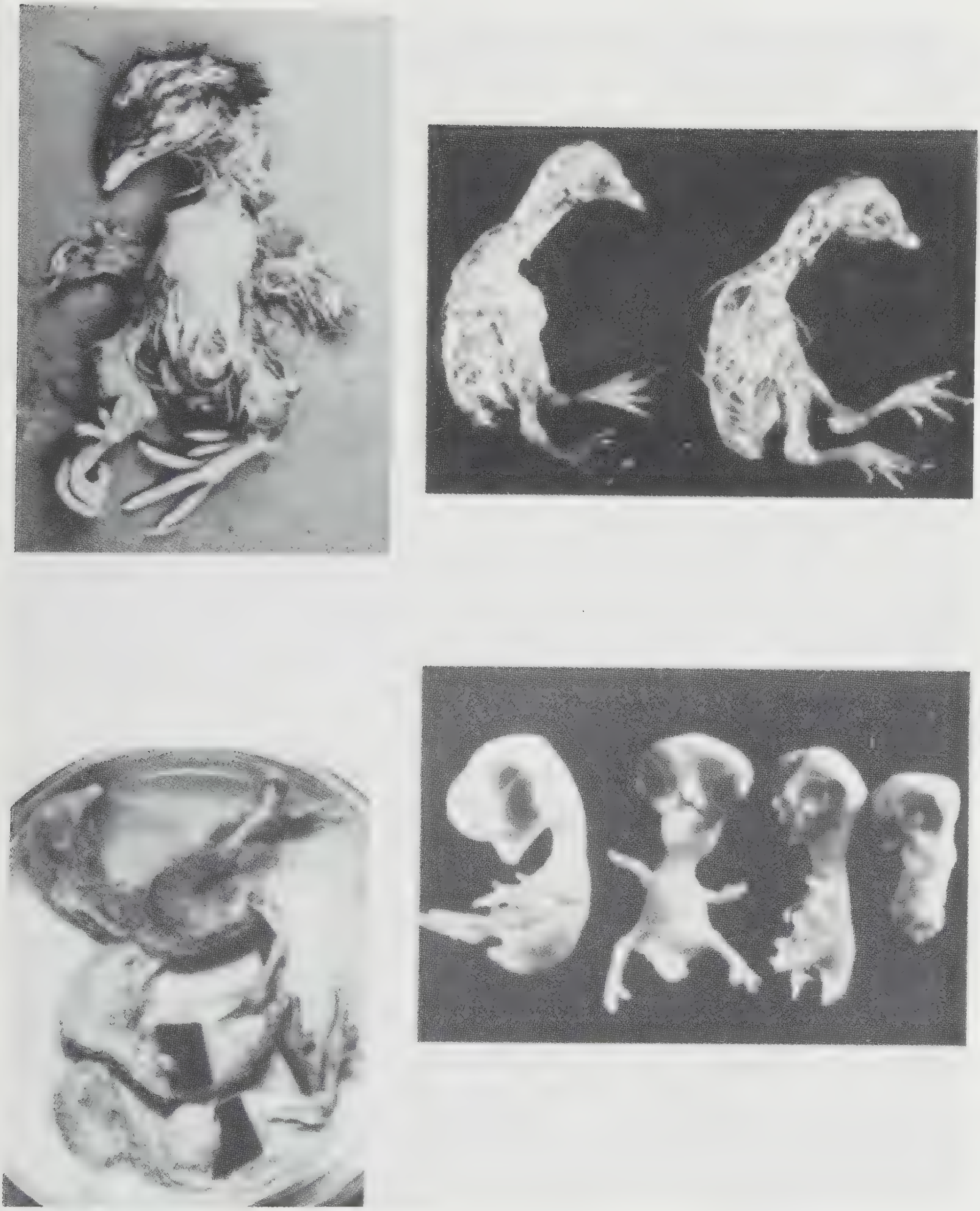

Figure 11.1. Polydactylous and hypodactylous embryonic lethals. Upper left: diplopodia-5 embryo at 22 days of incubation (from Olympio et al., J. Hered. 74:343, copyright 1983 Am. Genet. Assoc.). Upper right: splitfoot and normal embryos at 18 days of incubation (from Abbott and Goetinck, J. Hered. 51:161, copyright 1960 Am. Genet. Assoc.). Lower left: comparison of completely wingless (upper), 'vestigial wingless' (middle), and normal (lower) embryos (from Waters and Bywaters, J. Hered. 34:214, 1943). Lower right: normal embryo at nine days of incubation compared to mild, intermediate, and severe expression of coloboma (from Abbott et al., J. Hered. 61:96, copyright 1970 Am. Genet. Assoc.).

Red flock, was the first and only total amelia mutant ever reported in the chicken. It was possible to distinguish affected embryos as early as four days of incubation. Their limb buds had not developed as had the normals, and they were actually in stages of resorption. The limbs did not develop because there was no pectoral apical ectodermal ridge, but the pelvic girdle did develop fully. The upper beak was also affected, being one-half to one-third the length of the lower beak. The lungs and kidneys were well developed in this mutation, and two chicks that were helped out of the shell lived for a short while. Normally the homozygotes survived to hatching time, and then because of lack of legs were unable to break out of the shell.

The ratio from matings of known carriers (145:43) adequately demonstrated the simple autosomal recessive nature of this trait. Prahlad et al. (1979) assigned the gene symbol *am*, for amelia, to this trait. However, because *am* had previously been assigned to the muscular dystrophy trait, a new symbol (*ll*) was designated for limbless (Somes, 1984).

Stumpy (*stu*). An extremely hypodactylous mutant called stumpy was briefly reported by Abbott et al. (1966). This mutant appeared in the F_3 generation of a New Hampshire x Cornish mating and was subsequently determined to be inherited as an autosomal recessive. Eventually the gene symbol *stu* was assigned to the mutant (Somes, 1980).

Development does not progress beyond the sixth or seventh day of incubation at which time this condition results in death. At this stage, limb buds are only conically-shaped stumps with cartilaginous formation of the long bones confined to the femur and parts of the tibia and fibula. Other features include reduced eye size, poor vascularization, and hemorrhages in the brain, mesonephros, spleen, and liver. Limb mesenchyme transplantation studies indicated that the mutant mesenchyme cells controlled the phenotypic expression of this trait (Bishop-Calame and Abbott, 1967).

Coloboma (*cm*). A mutational defect with an enormous range in expression was reported from a White Leghorn source by Abbott et al. (1970) and given the name coloboma (Figure 11.1). The coloboma gene seems to act during the precartilaginous stage of development when mesenchymal condensations are forming in future cartilaginous areas. As a consequence of this, cartilage may be missing, reduced or deformed. Expression of this condition was arbitrarily subdivided into three grades of severity by the authors, and the time of mortality roughly correlated with the degree of expression. The mildest expression was lethal at the end of incubation, with the defect being restricted primarily to the beak where proximolateral parts of the maxilla were missing. Wings were slightly reduced, as was body size. Legs and toes were normal or lacked only the hallux. A few of this type may even have hatched. By contrast, the severest form affected all parts of the embryo. The skull and face were extremely deformed with the eyes displaced forward and the beak as only a rudiment. Wings were usually absent or reduced to spikes. Legs were less affected, but they were reduced in length because of the absence of distal bones and they were twisted. About one-third of these extreme embryos died between the fifth and sixth days of incubation, while the rest died between 13 and 16 days. An intermediate expression of this condition affected all the same cartilaginous elements but to a lesser degree. The time of lethality for this grouping was generally later, a few at 5-6 days and 13-14 days of incubation but the majority at hatching time.

Genetic studies showed that the coloboma trait was due to a single sex-linked recessive gene (Abbott et al., 1970). The original carrier male produced 25.1 percent mutant embryos, and the proportion produced by his proven carrier sons was 23 percent. All of the mutant embryos that were sexed were females. The authors assigned the gene symbol *co* to this trait, but because this symbol was already designated to a feather pigmentation gene, a new symbol (*cm*) was assigned (Somes, 1980).

CHONDRODYSTROPHIC EMBRYONIC LETHALS

The term chondrodystrophy is commonly used in reference to various conditions involving abnormal development of the limb long bones. In the chicken these would include manganese deficiency micromelia (Lyons and Insko, 1937), the various achondroplastic conditions described in Chapter 7, and the so-called sporadic as well as the known recessive inherited embryonic chondrodystrophies. Early genetic studies of chondrodystrophy concluded that these embryonic abnormalities did not have a genetic basis and were of a sporadic type (Dunn, 1927; Hutt and Greenwood, 1929; Munro, 1932). However, since those early studies, five single gene autosomal, one sex-linked, and one two-gene inherited forms of embryonic chondrodystrophy have been reported. The five single gene autosomal mutants have not been tested against each other for allelism and thus it is not assured that these represent five independent loci. These seven chondrodystrophic embryonic lethals are listed in Table 11.5.

Chondrodystrophy (*ch*). The first of these was reported by Lamoreux (1942) and further studied by Landauer (1965a). Lamoreux's chondrodystrophy varied considerably in phenotype. Extreme expression consisted of shortened and bent tibia, with wing bones being less affected (Figure 11.2). Short lower and curved upper mandibles contributed to a parrot-beak appearance. A modified expression ranged all the way to embryos with only a slight degree of beak abnormality and normal extremities. Landauer (1965a) was able over a period of nine years to select two divergent populations; in one line the incidence of extreme embryos rose to 95.7 percent, and in the other line the incidence of modified embryos rose to 77.8 percent. Most chondrodystrophic embryos died during the last days of incubation. A few modified embryos hatched but they seldom lived beyond a few weeks. Genetic studies showed this condition to be due to a single autosomal recessive gene to which Lamoreux (1942) assigned the gene symbol *ch*.

Cartilage from *ch/ch* embryos was shown to contain a normal complement of chondroitin sulfate (Mathews, 1967). Landauer (1965b) showed in a phenocopy-producing study that the multiple modifying genes which tend to suppress the *ch/ch* genotype, as in

Table 11.5. Chondrodystrophic embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Chondrodystrophy-Lamoreux	*ch*, Ch^+	Lamoreux (1942)
Micromelia-Asmundson		Asmundson (1942)
Micromelia VII	*mm-VII*; $Mm\text{-}VII^+$	Bernier (1951)
Micromelia-Kawahara	*mm-K*, $Mm\text{-}K^+$	Kawahara (1956)
Micromelia-Abbott	*mm-A*, $Mm\text{-}A^+$	Landauer (1967)
Nanomelia	*nm*, Nm^+	Landauer (1965c)
Sex-linked recessive		
Sex-linked chondrodystrophy	*chz*, Chz^+	Mann (1963)

the modified type, also greatly reduced the responsiveness of Ch^{+}/ch embryos to injections of 6-aminonicotinamide (6-AN). This was contrary to the effect of 6-AN on embryos heterozygous for the micromelia-Abbott gene.

Micromelia-Asmundson. Asmundson (1942) reported on a condition that he called micromelia, saying that it superficially resembled the Cornish short-legged lethal described in Chapter 7. This micromelia is now referred to as micromelia-Asmundson to distinguish it from the other micromelias. Micromelia-Asmundson also differs from other micromelias in that its inheritance results from the action of two autosomal recessive genes. The ratios from Asmundson's genetic studies (716:47) were a close fit to an expected 15:1 ratio. These micromelic embryos had very short and thickened extremities and their skulls were usually vaulted, with the upper and lower beaks shortened and deformed. Death was during the last days of incubation. An interesting observation was that the percentage of bone ash was higher in the micromelic long bones than in the normals, so that the bone matrix was reduced but not the ash content.

Micromelia VII (*mm-VII*). Bernier (1951) described a condition he called micromelia VII which was inherited as an autosomal recessive. It was very similar to other types of micromelias except for the fact that affected embryos had normal beaks and microphthalmic eyes. The gene symbol *mm-VII* was given to this trait (Somes, 1980).

Micromelia-Kawahara (*mm-K*). Yet another micromelic mutant was briefly reported by Kawahara (1956) in a White Leghorn flock. The long bones of both the legs and wings were very short, the beak was shortened, and the body was soft because there was very little calcium in the bones. There was a large quantitity of magnesium in the micromelic bones. The amniotic and allantoic fluids were retained and were highly viscous. A simple autosomal recessive gene substitution explained the inherited basis of this trait to which the name micromelia-Kawahara (*mm-K*) has been applied (Somes, 1980).

Micromelia-Abbott (*mm-A*). A lethal micromelia-like phenotype was discovered by Abbott at the University of California in a New Hampshire flock, but a full description of it has not been published. This mutant has been maintained since 1958 at University of Connecticut by outcrosses to White Leghorns. Embryos develop a severe form of micromelia, a parrot-like beak, and have retarded feather development, and hemorrhagic skin (Quintner and Goetinck, 1981). It behaves in matings as an autosomal recessive inherited trait. Embryonic death is generally early in the third week (Landauer, 1967). Landauer (1965b, 1967) referred to this mutant as California micromelia and gave it the gene symbol mm^{A}. However, it is more commonly referred to now as micromelia-Abbott and the gene symbol has been changed to *mm-A* to infer that it is not an allele of some of the other micromelias.

Micromelia-Abbott has been used in several experiments at University of Connecticut. In a phenocopy study with 6-aminonicotinamide (6-AN) to produce micromelia and parrot beak phenocopies, it was found that a single dose of the *mm-A* gene in a normal embryo made it much more responsive to the 6-AN treatment (Landauer, 1965b). Ultrastructural studies showed a reduced number of matrix granules, and a reduction in Golgi vacuoles and in the scalloping of the cell surface of the mutant chondrocytes (Sawyer and Goetinck, 1979). Biochemical analysis of cartilage proteoglycan revealed a reduction in the quantity but not the quality of sulfated proteoglycan in the mutants, which varied with the cartilaginous rudiment and developmental age, being more severe at earlier developmental stages. It was further suggested that this reduction may be due to a reduced availability of the xylosylated protein backbone for proteoglycan sulfate (Quintner and Goetinck, 1981).

Sex-linked chondrodystrophy (*chz*). Mann (1963) reported a chondrodystrophy-like mutant with a sex-linked mode of inheritance. He assigned the gene symbol *chz*, for Z chromosome chondrodystrophy, to what is now known as sex-linked chondrodystrophy.

Nanomelia (*nm*). In a deliberate attempt to uncover lethal mutant genes by disrupting their 'epigenetic armor', Landauer (1965c) was able to recover from the descendants of a Buff Cochin x White Leghorn cross deformed embryos with extraordinarily hypoplastic extremities, brachycephaly, and parrot-like beaks (Figure 11.2). Because the extremities were less abnormal than the phocomelia of creeper homozygotes, but were obviously more extreme than most micromelics, he designated this lethal mutant as nanomelia, and assigned the gene symbol *nm* to it when its autosomal recessive mode of inheritance was determined. Other interesting features of nanomelia were heart hypertrophy in *nm*/*nm* individuals, and an average of 6.3-10 percent reduction in length of long bones of Nm^{+}/nm individuals. As with other chondrodystrophic lethal mutants, the time of death was after 18 days of incubation.

An extensive literature now exists on the biochemical basis of nanomelia. The cartilage of nanomelic embryos contains only ten percent of the normal amount of chondroitin sulfate, whereas skin content is normal (Mathews, 1967; Fraser and Goetinck, 1971). Nanomelic chondrocytes have the potential to synthesize chondroitin sulfate when supplied with a suitable acceptor, and this chondroitin sulfate is normal in molecular size and composition (Stearns and Goetinck, 1979). Nanomelic chondrocytes also differ in their cAMP levels and their somatomedin sensitivity (Bourret et al., 1979). All chondrocytes, whether of mesodermal or ectodermal anlagen, are affected (McKeown and Goetinck, 1979), with the major cartilage proteoglycan species (PGS-I) being reduced to levels of 1-2 percent of normal (Pennypacker and Goetinck, 1976; Palmoski and Goetinck, 1972). Even this small amount of PGS-I synthesized by the nanomelic embryos is abnormal in its characterization (McKeown-Longo and Goetinck, 1982). The minor cartilage proteoglycan species (PGS-II) (Palmoski and Goetinck, 1972), link protein (McKeown-Longo et al., 1983), type II collagen (Pennypacker and Goetinck, 1976), precartilaginous tissue (Sawyer and Goetinck, 1981), and noncartilaginous tissues such as skin fibroblasts (Goetinck and Royal, 1976) are not affected by the *nm* gene. Reduction of PGS-I was shown to be due to a deficiency in the production of core protein (Argraves et al., 1981), and yet nanomelic chondrocytes do synthesize a smaller glycoprotein but it does not undergo the normal posttranslational processing that is characteristic of normal cartilage core protein (O'Donnell et al., 1988). Faulty transcriptional regulation of the PGS-I core protein gene was suggested as being the cause because there was no indication that the core protein gene itself is missing (Stirpe et al., 1987).

FACIAL EMBRYONIC LETHALS

The eight facial embryonic lethals are listed in Table 11.6. All of these mutants are autosomal recessive.

Duck beak and Donald Duck (*dck, dd-2, dd-3*). A similar type of curled beak abnormality has been reported three times. Broekhuizen and van Albada (1953) reported

Table 11.6. Facial embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Duck beak	*dck, Dck*$^+$	Broekhuizen and van Albada (1953)
Donald Duck-2	*dd-2, Dd-2*$^+$	Abbott and Lantz (1967)
Donald Duck-3	*dd-3, Dd-3*$^+$	McGibbon (1973)
Missing maxillae	*mx, Mx*$^+$	Asmundson (1936)
Missing mandible	*md, Md*$^+$	Marble et al. (1944)
Missing upper beak	*mub, Mub*$^+$	Silversides et al. (1982)
Ectrodactyly	*ec, Ec*$^+$	Abbott and MacCabe (1966)
Perocephaly	*per, Per*$^+$	Landauer (1956a)

what they called duck beak in the Barnevelder breed. Abbott and Lantz (1967) called it Donald Duck-(2) beak in their New Hampshires, and McGibbon (1973) called it Donald Duck-(3) in his White Leghorns. In all these instances the phenotypes were quite similar; the maxillae of normal length curved upward and a shortened mandible curved downward (Figure 11.2). There was usually some variability in the extent of the beak abnormality. Abnormal embryos could be recognized as early as 8-10 days of incubation. Because of the nature of the condition, most embryos died just before or during hatching; a few chicks were able to hatch but most died by seven days of age, although one managed to live for about seven weeks (Broekhuizen and van Albada, 1953).

Genetic tests showed in each case that these beak abnormalities resulted from a single autosomal recessive gene. The gene symbols *dck* (Broekhuizen and van Albada, 1953), *dd-2* (Abbott and Lantz, 1967), and *dd-3* (McGibbon, 1973) have been assigned to each of these loci. Since none of these three mutants has been tested against each other, it is possible that they may occupy the same locus.

Missing maxillae (*mx*). Asmundson (1936) found a defect of the beak in a flock of inbred White Leghorns that was lethal when embryos were homozygous for it. In these embryos, the maxillae of the upper mandible were either absent or greatly reduced in size. The premaxillae were usually present and normal, but were reduced in size. The nasals, other facial bones, and the eyes were also sometimes reduced in size. The upper beak was frequently bent to one side. Heterozygous embryos appeared to be normal. This condition was distinguishable by at least the 12th day of incubation. Embryos were viable up to hatching time, but they could not pip because of the deformed beaks; one chick hatched out of 58 affected individuals, and seven were removed from the shell, but all died within one week.

A single autosomal recessive gene was found to cause this mutant condition, when a ratio of 173:58 was obtained from matings of heterozygotes. Asmundson (1936) called the condition abnormal upper mandible, while Hutt (1949) called it abnormal maxillae, missing maxillae, and amaxilla, and assigned to it the gene symbol *mx*.

Missing mandible (*md*). This lethal condition, which affects the head area of embryos, was reported by Marble et al. (1944) in a White Leghorn strain. The lower mandible was reduced to only a vestige of its normal size, while the upper mandible rarely exceeded two-thirds of its normal size. In some of the embryos, the upper mandible either

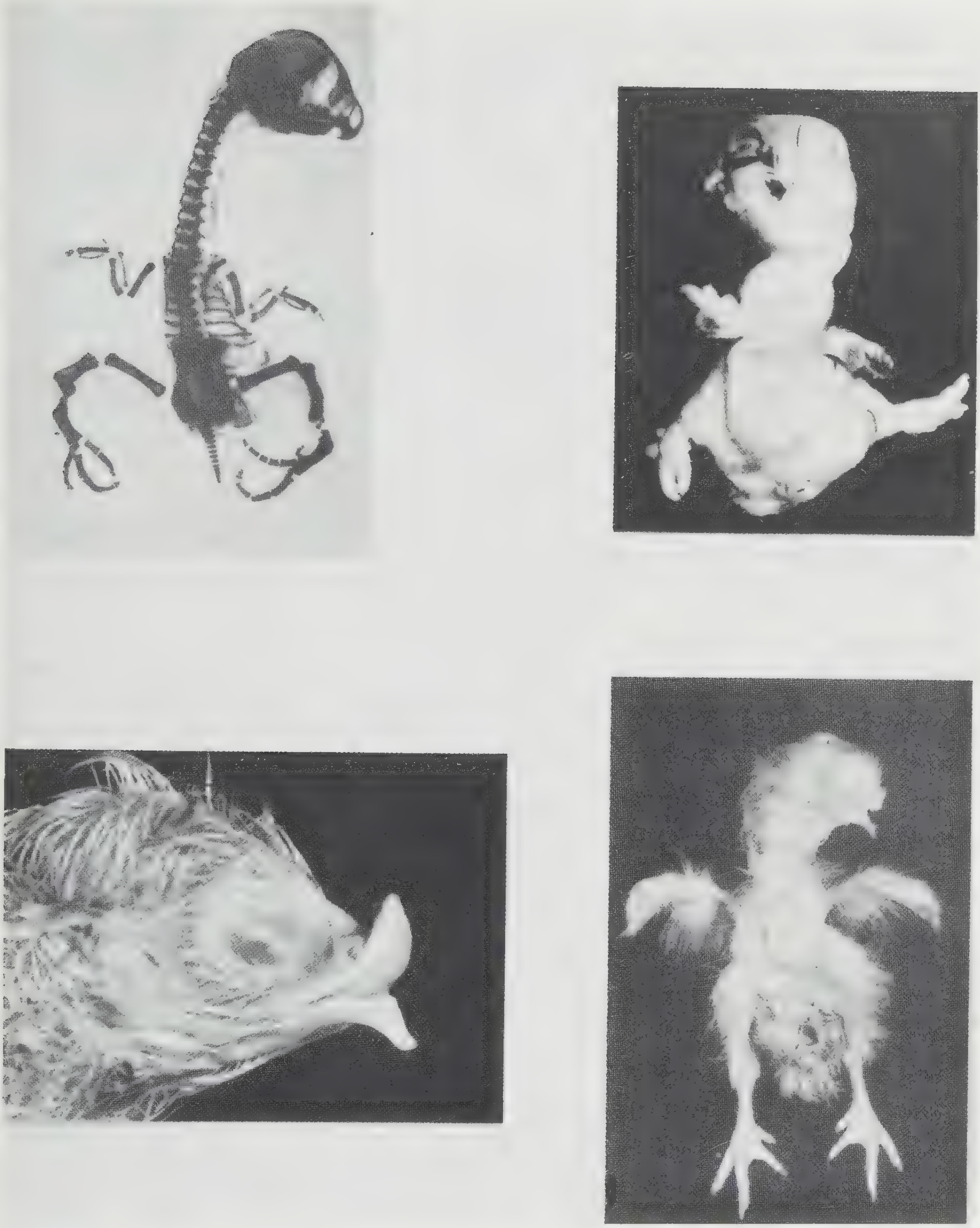

Figure 11.2. Chondrodystrophic and facial embryonic lethals. Upper left: extremely chondrodystrophic embryo at 21 days of incubation, cleared and stained with alizarin (from Lamoreux, J. Hered. 33:277, 1942). Upper right: nanomelic embryo at 20 days of incubation, down removed to reveal head and limb deformities (from Landauer, J. Hered. 56:132, copyright 1965 Am. Genet. Assoc.). Lower left: Donald Duck embryo at 20 days of incubation (from Abbott and Lantz, J. Hered. 58:240, copyright 1967 Am. Genet. Assoc.). Lower right: ectrodactylous embryo at 18 days of incubation (from Abbott and MacCabe, J. Hered. 57:209, copyright 1966 Am. Genet. Assoc.).

pointed upward or curved over the front of the head. Frequently the eyes were affected, and there was always some degree of cerebral hernia. The condition was evident as soon as beak development began, but its lethal action was restricted to the end of incubation when embryos were unable to pip the shell.

The combined ratio in progeny from matings in three family groups of carriers (378:134) demonstrated the autosomal recessive nature of this trait. Hutt (1949) assigned the gene symbol *md*.

Missing upper beak (*mub*). A new condition, resulting in abnormal membranous bone formation and affecting the facial structure of domestic fowl, was described by Silversides et al. (1982). Only membranous skull bones were involved. The frontal bones were reduced in size and the upper jaw was reduced to just the stump of the beak. The lower jaw remained nearly normal. Part of the upper eyelid was always missing, and in severe cases both eyelid and nictitating membrane were absent. Time of death for this lethal was equally divided between 12 days of incubation and the end of incubation. Those alive at the end were unable to pip the shell. One affected chick did hatch without assistance and five others were helped out of the shell, but none survived beyond 11 days of age.

Heterozygote x heterozygote matings produced a ratio of 332:117. This result, along with reciprocal backcrosses to normals which produced all normals, verified the single autosomal recessive nature of this trait. The authors proposed *mub* as the gene symbol.

Ectrodactyly (*ec*). Abbott and MacCabe (1966) reported a very interesting lethal mutant that appeared among the progeny of a junglefowl x New Hampshire cross. They chose to name this new lethal syndrome ectrodactyly. When it was determined that it was inherited as a single autosomal recessive, they assigned the gene symbol *ec*. Ectrodactylous embryos were always missing the entire premaxilla and the palatine process, and frontal bones were pushed back at their point of union. This gave the upper beak a snout-like appearance (Figure 11.2). An unusual feature of ectrodactyly was that the phenotype differed depending upon the genetic background. When ectrodactylous embryos were also homozygous for the scaleless gene (*ec/ec sc/sc*) they had feet and leg defects in addition to the beak defects, varying from only a slight reduction of the hallux to a complete absence of legs. In all cases ectrodactylous embryos died between 17 and 20 days of incubation. Leg defects could be recognized by three days of incubation but beak defects were difficult to identify before the sixth day. Yee (1977) examined the facial development of this mutation and compared it with normal embryos using both light and electron microscopy.

Perocephaly (*per*). A mutation represented by a wide range of malformations of the head was described by Landauer (1956a) as perocephaly. Malformations ranged from a rudimentary upper beak to microcephaly. In succeeding steps, the eyes were transposed forward until they merged as a cyclopean eye (with the lower beak remaining normal), the single median eye was reduced in size or was only a pigmented spot, the proboscis disappeared, and finally there was no visible sign of nose, eyes, or mouth and the skull and brain were mere vestiges. Embryos with low-grade defects usually lived to hatching time but were unable to pip because of the beak defect. Most defective embryos died during the early part of the third week. As the incidence of perocephally increased in the study flocks, there was an associated increase in early embryonic mortality.

Percephalic embryos first appeared in the F_2 and later generations of crosses between recessive rumpless fowl and Silver Grey Dorking, Rhode Island Red and junglefowl. The initial incidence of the trait was 2-3 percent. This rose to about five percent and then 13 percent when all genotypes of all sources of origin were combined. The breeding history of these crosses made it clear that the perocephalic gene had been present in the recessive rumpless stock and probably in the White Leghorn stock from which it had been derived in a submerged condition. The mutant condition was completely suppressed by modifying genes, and it only began to show itself as the influence of the modifiers was diluted by outcrossing. It was suggested that suppression of the action of deleterious mutants may represent a common and important mechanism for equilibration and genetic self-defense. Despite all the modifier genes, one major recessive gene was believed to be responsible for this lethal complex, and it has been assigned the gene symbol *per* (Somes, 1980).

In three out of four breeding seasons, the incidence of perocephalic embryos increased significantly as the breeding season progressed. Experimental evidence (Landauer, 1957) suggested that this shift in mutant embryo frequency was due to lowering of the oxygen tension in the air surrounding the incubating eggs. It also demonstrated that some homozygous *per/per* chicks could develop into perfectly normal chicks.

MISCELLANEOUS EMBRYONIC LETHALS

Six miscellaneous embryonic lethals are listed in Table 11.7. Four of these are autosomal recessive and two are sex-linked recessive. Most are mutants which affect soft tissue.

Stickiness (*sy*). An unusual lethal factor, acting during the last four days of incubation, segregated from a mating between Barred Plymouth Rock and Rhode Island Red. This was one of the earliest reported lethals. Byerly and Jull (1932) labelled it stickiness because the amniotic and allantoic fluids were not absorbed and were unusually viscous at the time of death. Sticky embryos could usually be distinguished by 11 days of incubation, but there was a greater degree of certainty in classifying older embryos. Affected embryos were smaller in size, with distended abdomens due to a large amount of unabsorbed yolk. They had swollen throats and varying amounts of generalized edema. Their bones were extremely soft, and about a third of them had tibial bending typical of chondrodystrophy.

Subsequent matings indicated that the mutant gene had originated from the Barred Plymouth Rock breed. *Inter se* matings of tested carriers yielded a ratio of 545 normal to 169 sticky embryos. When carriers were outcrossed, only normal chicks were produced. This was conclusive proof that this trait was inherited as an autosomal recessive and that it

Table 11.7. Miscellaneous embryonic lethals.

Inheritance and trait name	Gene symbol	Reference
Autosomal recessive		
Crooked neck dwarf	*cn*, Cn^+	Asmundson (1945)
Dorking lethal	*dl*, Dl^+	Landauer (1959)
Open breast syndrome	*obs*, Obs^+	Fiser et al. (1972)
Stickiness	*sy*, Sy^+	Byerly and Jull (1932)
Sex-linked recessive		
Late sex-linked lethal-Upp	*lsl*, Lsl^+	Upp and Waters (1935)
Liver necrosis	*ln*, Ln^+	Cole and Jones (1972)

was not due to environmental or incubational factors, or peculiarity of the eggs of particular hens. Hutt (1949) assigned the gene symbol *sy* to this lethal condition.

Open breast syndrome (*obs*). Fiser et al. (1972) reported a condition that they called open breast syndrome, which specifically affected the sternal crest area. The sternal crest (keel) and caudolateral processes were not properly developed and remained in a vestigial form. The breast muscles atrophied because they lacked a site of attachment. This condition was lethal to all affected embryos and none was ever observed to hatch. It could be detected as early as five days of incubation, but only about four percent died during the first week of incubation. The great majority (70 percent) died during days 18-20 and another 26 percent during the second week of incubation.

Breeding tests between known carriers gave ratios (1147:347) that were not significantly different than would be expected for the segregation of a single autosomal recessive gene. The gene symbol *obs* has subsequently been assigned to this locus (Somes, 1980).

Dorking lethal (*dl*). Very few lethal genes cause death during the second week of incubation, but Landauer (1959) reported on just such a trait from a Silver Grey Dorking mating. In this case the defective embryos were readily identifiable at seven days of age, with major mortality occurring during the eighth day and some during the early part of the ninth day. A short neck was the most striking feature of this lethal condition, and because of this Landauer called the condition short-neck. However, over the years it has become known as the Dorking lethal. Other features of affected embryos include shortened mandible and tail, and a 15-25 percent reduction in body weight. A nonskeletal feature of this trait was an increase in eye pigment intensity.

Extensive matings involving 96 carrier females and 58 noncarrier females produced results that agreed well with the hypothesis that this short-neck lethal was the result of a single autosomal gene. At a much later date, the gene symbol *dl* was designated (Somes, 1980).

Crooked neck dwarf (*cn*). The crooked neck dwarf lethal was reported by Asmundson (1945) to have been derived from a New Hampshire flock. Embryos died at 20-21 days of incubation. They had a short upper beak, a crooked neck, a reduced sternum with the keel and lateral processes of the sternum being absent, and a slight reduction of the long bones (Figure 11.3). The breast muscles were absent or greatly reduced, and leg muscles and overlying tissues were also reduced. Other pleiotropic abnormalities (Pun, 1954) consisted of crooked spinal column, spindly legs, curled toes, and esophageal atresia. The muscle reductions caused a weight reduction and thus the dwarf-like state, the crooked neck, and a rigidness of the hocks. Body weight was normal up to 12 days of development, but it slowed down between 13 and 16 days and ceased increasing after 17 (Asmundson, 1945).

Several histological studies of the various affected tissues have been reported: esophagus (Allenspach, 1966), trachea and thymus (Allenspach, 1970), muscles (Rosenberg, 1947; Elson, 1972; Wick and Allenspach, 1978), nerves (Harris, 1966; Rosenberg, 1947), and skin (Elson, 1972). Mathews (1967) examined the chondroitin sulphate content of *cn/cn* embryo collagen and found it to be similar to that of normal embryos.

A series of studies has indicated that muscle pattern development and subsequent pattern maintenance is a two-step process, and that the *cn* gene only interferes with the second step (Kieny et al., 1983; Mauger et al., 1983). The splitting of the initial muscle masses and

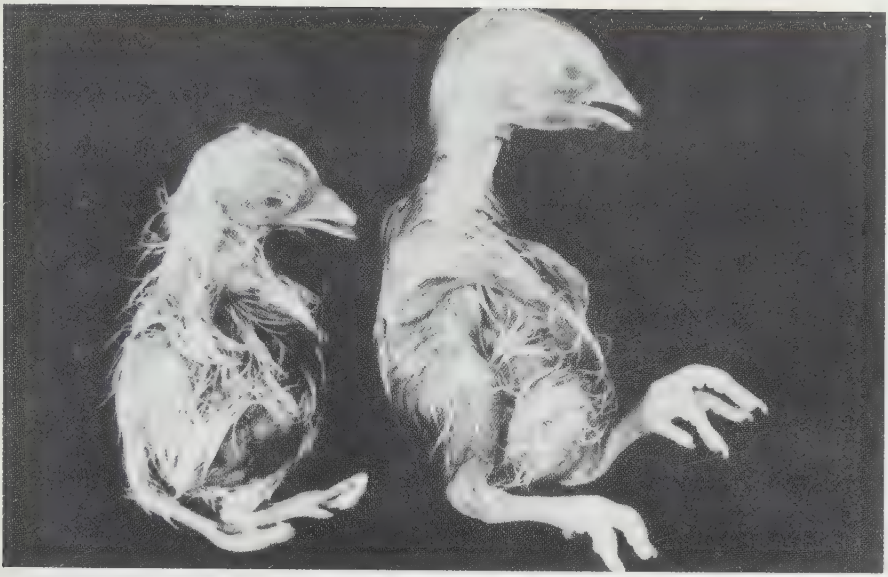

Figure 11.3. Crooked neck dwarf (left) and normal (right) embryos (from Asmundson, J. Hered. 36:174, 1945).

the spatial arrangement of the muscle progresses up to 7.5 days of development, and then is followed by a secondary fusion of individual muscles into unorganized muscle tissue surrounding the tibiotarsus and fibula. Thus the *cn/cn* muscular dysgenesis results from a defect of somitic myogenic cells which prevents the muscle pattern from becoming a stabilized definitive structure. Muscle connective tissue later becomes disorganized (Mauger et al., 1983) although the skin is not affected (Mauger et al., 1984). The spatial and temporal distribution of extracellular matrix components, type I and II collagen, fibronectin, and laminin, of *cn/cn* developing muscle differs from that of normals (Mauger et al., 1984). The action of the *cn* gene is limited to skeletal muscle. Cardiac and smooth muscle show structure and development similar to that of normals (Kieny et al., 1988).

This unusual and much studied lethal trait is inherited as an autosomal recessive. Among the progeny of heterozygous parents there were 103 crooked neck dwarf embryos from a total of 400 chicks and embryos (Asmundson, 1945), which is extremely close to the expected 3:1 ratio. Hutt (1949) applied the gene symbol *cn* to this syndrome.

Phenocopies of the crooked neck condition have been produced using nicotine sulphate. Cn^+/cn embryos were much more responsive to it than were Cn^+/Cn^+ embryos (Landauer, 1960). It has been shown that 3-acetyl pyridine induces hypoplasia of leg muscles in normal chicks, closely paralleling the changes in crooked neck embryos (Hermann et al., 1963).

Late sex-linked lethal-Upp (*lsl*). Upp and Waters (1935) reported what was probably the first example of a sex-linked lethal. Their data were obtained from sexing dead embryos of an inbred White Leghorn stock. They recorded 331 dead male and 586 dead female embryos from ten sires, all of which were presumed to be heterozygous for a sex-linked recessive gene. Death had occurred late in the incubation period. No abnormalities or causes of death were described in the report. It is proposed that the gene symbol *lsl* be given to this condition.

Liver necrosis (*ln*). Cole and Jones (1972) briefly reported a rather interesting sex-linked trait that appeared in one of their White Leghorn flocks. This condition expressed itself as a necrosis of the liver. It could be grossly recognized as early as 12 days of incubation. The majority of affected females died near the end of the incubation period; a few hatched that were free of symptoms, but on examination at three days of age they were shown to have liver lesions.

Approximately half the daughters of the original three heterozygous males died, and of 41 sons that were tested, 23 proved to be heterozygous. This unusual lethal condition was inherited as a single sex-linked recessive and the gene symbol *ln* was assigned to it (Cole and Jones, 1972). Additional generations of breeding seemed to change its phenotypic expression through the influence of multiple modifying genes so that eventually only 68 percent of the expected numbers of *ln*/- females showed liver lesions.

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Chapter 12. MUTATIONS AND MAJOR VARIANTS IN TURKEYS

T.F. Savage

The literature pertaining to single gene variation in the turkey is scanty in comparison with that for the chicken. Some of the reasons for this paucity of information are the relatively high cost of turkey research, the low reproductive rate of the species, and its relatively limited agricultural utilization. With increased consumer demand for turkey and turkey products, research interest in the turkey is expanding and perhaps more contributions to basic genetic knowledge will result in the years ahead.

Domestic turkeys are descendants of the true wild turkey (*Meleagris gallopavo*) of North America (see Chapter 1). Two subspecies made major contributions to modern domestic turkeys; these were the Mexican (*M. g. gallopavo*) and the eastern subspecies (*M. g. silvestris*). Wild-type genetic traits have been retained in some domestic stocks, particularly in the Standard Bronze.

PLUMAGE COLOR AND PATTERN VARIANTS

There are presently nine documented single gene loci that directly influence plumage color or patterns in turkeys (Table 12.1). Of these, seven are autosomal, two are sex-linked, and three loci have multiple alleles.

Bronze or wild-type. Down color in the day-old bronze poult consists of the following. The poult's head has a light brown base color that is mottled and contains three parallel streaks of dark brown. The upper body parts are streaked and mottled with dark brown and black pigment. The undercolor of the dorsal down is reddish-yellow. The ventral body surface from the neck to the tail is yellowish-white with a medium-grey undercolor.

The typical adult bronze turkey has an iridescent red-green bronze color on the surface of the feathers covering the neck, breast, wing bows and fronts, secondary coverts, and the anterior portion of the back. The remaining feathers on the back are bordered with a band of copper-colored bronzing having a black-pigmented edge. The proximal third of the tail coverts and main tail feathers are black with pencilled bands of brown at the distal end which terminates in a wide band of copper-colored bronzing, a narrow black band, and a concluding edge of white. The flight feathers, primary coverts, and pinions are uniformly marked with alternating dull black and white bars. The feathers of the lower thighs are black with a grey-white edging, and the abdomen is dull black. In the bronze female, there is a white edge to the black bars on the back, wing bows and coverts, and breast. In both males and females, the beak is reddish-brown, the eyes are dark brown, the beard is usually black, and the skin is white (Marsden and Martin, 1945).

Black (*B*) and black-winged bronze (b^l). There is a series of three alleles which are responsible for black, bronze, and black-winged bronze plumages. The order of dominance is $B > b^+ > b^l$.

The gene causing black plumage is autosomal and dominant to the other alleles at this locus and is designated with the symbol B (Asmundson, 1945a). Poults have black down without stripes on the dorsal body surface. Occasional white pigmentation is present in the tips of the primary and secondary flight feathers. The poult has a band of yellow-colored down that surrounds both sides of the face and the eyes. Yellow down is present on the ventral body surface. Some of the black pigmentation extends into the legs but the feet are usually pink. In adults, all of the feathers are black although there may be white flecking in the flight feathers.

The black-winged bronze phenotype was described by Asmundson (1939) as a nonbarred condition of the flight feathers in otherwise bronze birds. This color pattern is also referred to as Crimson Dawn. The autosomal recessive gene responsible for this condition was originally symbolized as l and is now designated b^l. In a subsequent study, Asmundson (1945a) determined that black-winged bronze was a third allele present at the b^+ locus with the allelic hierarchy being $B>b^+>b^l$. The b^l gene has been shown to have epistatic effects when in combination with genes responsible for red, white, and the two slate plumage color phenotypes.

The down pattern of the black-winged poult is similar to that of the bronze or wild-type, but intensity of the brown and black pigmentation is reduced so that the down is nearly white. The typical bronze pattern is present in adults of both sexes, but the primary flight feathers are black without barring; the secondary flight feathers are also plain black and they have white tips. In females with this feather pattern, many of the shoulder feathers are white and grey.

White (c) and grey (c^g). This autosomal locus was initially identified as W (Robertson, 1929) but was renamed as the color or C locus (Robertson et al., 1943). It has a profound effect on plumage color expression. Three alleles are known at this locus including the genes responsible for wild-type color, grey, and white plumages. The order of dominance is $C^+>c^g>c$.

The dominant allele at this locus, C^+, permits expression of other color loci.

A second allele, c^g (Nestor and Renner, 1979), is recessive to C^+ and results in grey coloring (Buss, 1960). The day-old grey poult is whitish-grey with a black stripe of varying intensity on the back. The ventral surface is yellow-colored. In the adult, the feathers are white with black barring, except for the flight feathers which have a smearing of black throughout instead of the usual black and white bars. The tail feathers are white with brown or black pigmentation, and there is a black bar at the end with a white edging.

The third allele at this locus, c, is completely recessive. In the homozygous condition it prevents the expression of color by other loci (Robertson et al., 1943). This absence of color does not infer an albinotic condition. Down color of the day-old poult is yellow with buff on the head and tan stripes on the back. There is an interaction between c and the B locus; poults that are c/c and B/B, B/b^+, B/b^l or b^l/b^l will have white down but without the buff coloration. In the recessive white adult, feathers are completely devoid of color pigmentation. Nestor and Renner (1979) reported that the c allele exhibited pleiotropy when contrasted with the c^g allele; recessive white males had significantly lower semen volumes and sperm concentration than their homozygous grey counterparts.

Red (r). The gene for red plumage, as found in the Bourbon Red, results in a reddish-brown coloration in the day-old poult and in feathers of the adult. In the day-old poult there is a dark spot on the back of the reddish-brown head. The down above the eyes varies from brown to tan, while below the eyes and extending to the throat, breast, and thighs the down is creamy yellow. On the back, the down is yellow to light brown with three medium to

Table 12.1. Listing of mutations and major variants in turkeys.

Trait and gene symbol		Trait and gene symbol	
Variations in plumage color and pattern			
Black, black-winged bronze	B, b^{l}, b^{+},	Palm	p, P^{+}
Grey, white	c^{g}, c, C^{+}	Spotting	sp, Sp^{+}
Red	r, R^{+}	* Narragansett, imperfect albinism	n, n^{al}, N^{+}
Dominant slate	D, d^{+}	* Brown	e, E^{+}
Recessive slate	sl, Sl^{+}	Faded bronze	fb, Fb^{+}
Variations in feathers and skin			
Hairy	ha, Ha^{+}	Knobby down	kn, Kn^{+}
* Late feathering	K, k^{+}	Dermal melanosis	m, M^{+}
Naked	na, Na^{+}		
Embryonic lethal mutations			
Chondrodystrophy	ch, Ch^{+}	Ring lethal	rl, Rl^{+}
Chondrodystrophy-m	$ch\text{-}m, Ch\text{-}m^{+}$	Shortened long bones	s, S^{+}
Crooked neck dwarf	cn, Cn^{+}	Short-spined	sh, Sh^{+}
Hemimelia	hm, Hm^{+}	Swollen down plumules	sdp, Sdp^{+}
Micromelia-like	mm, Mm^{+}		
Neurological mutations			
Congenital loco	lo, Lo^{+}	* Bobber	bo, Bo^{+}
* Vibrator	vi, Vi^{+}		
Metabolic mutations			
Binucleated erythrocytes	bn, Bn^{+}	Hereditary glaucoma	ga, Ga^{+}
Bowed hocks	bh, Bh^{+}	Muscular dystrophy	dy, Dy^{+}

* Sex-linked. All other loci listed are autosomal.

dark brown stripes. The wings exhibit a progression from the wing fronts to the tips of dark brown to cream.

In most feathered areas of the adult, excluding the flight, tail, and breast feathers, the plumage color is dark brown to red with occasional black flecking. The flight feathers are solid white. The tail feathers have a faint white bar near their ends. In males the breast feathers are edged in black, and in females they have a white border.

The gene responsible for red plumage color is autosomal recessive and it is at a different locus than bronze (Robertson, 1929). The gene symbol is r. The wild-type allele R^{+} is incompletely dominant and results in bronze (R^{+}/R^{+}) and bronze-red (R^{+}/r) phenotypes. Robertson et al. (1943) and Asmundson (1945a) have described in tabular form some of the phenotypic colors resulting from epistatic interaction of this locus with several other color-determining loci in the turkey.

Slate (*d, sl*). This plumage color, varying from light grey to blue-grey, results from either of two different autosomal mutations. One is dominant and one is recessive. Both are independent of and epistatic to the black color locus and its alleles.

Dominant slate. Robertson et al. (1943) described the dominant form of slate plumage and adopted the gene symbol D. The dominant slate gene reduces black pigmentation to a uniform slaty-blue throughout the plumage, but it has little effect on red pigmentation.

Dominant slate is actually incompletely dominant, as seen in the heterozygote which has a color pattern exhibiting black pigment. When the bird is genotypically bronze and homozygous for slate (D/D), a slaty-blue individual with barred wing and tail feathers results. Robertson (1929) described other slate color variants that resulted from interaction with other color loci.

Recessive slate. Asmundson (1940) described the second source of slate color as the expression of a recessive gene (*sl*). The dominant allele Sl^+ was responsible for nonslate or bronze. Two phenotypes of slate were possible when the *sl* gene interacted with the red locus; slate color with barred flight and pencilled tail feathers occurred when the genotype was R^+/R^+ or R^+/r and sl/sl; lilac, a variant of slate with nearly white flight feathers and unpencilled tail feathers, resulted when both loci were homozygous recessive (Asmundson, 1945a).

Palm (*p*). Palm plumage color, as seen in the Royal Palm variety, was described by Marsden and Martin (1945) and by Asmundson (1945a). The autosomal palm locus contains two alleles. The dominant allele (P^+) expresses the wild-type or bronze phenotype, while the recessive *p* is responsible for the palm phenotype. The palm down color is light yellow with or without black stripes on the back. The adult plumage consists primarily of silvery-white feathers with iridescent black bars and white tips. The primary flight feathers are dull black and the secondaries contain decreasing amounts of black pigment.

Spotting (*sp*). Asmundson (1955) described a variant of black plumage color as spotting or Nebraskan. This color pattern was the expression of an autosomal gene (*sp*) that was recessive to wild-type or bronze. The down in day-old poults was white with one or more brown spots on the back of the head. Adult plumage consisted of white feathers with black pigment dispersed in all feathers. There was a greater deposition of black in the feathers of the neck, back, and parts of the wings, but there was no barring in the flight feathers.

The *sp* gene appears to function by suppressing or delaying deposition of black feather pigment in the young bird; in the adult, brown pigmentation is not visible. It has been postulated that the turkey *sp* gene is analogous to the autosomal recessive red-splashed white gene (*rs*) in chickens.

Narragansett (*n*) and imperfect albinism (n^{al}). The narragansett locus is sex-linked. It consists of three alleles which are responsible for bronze, narragansett, and imperfect albino phenotypes. The order of dominance is $N^+ > n > n^{al}$.

Narragansett. The narragansett plumage color, as seen in the Narragansett variety, is a modification of bronze or wild-type in which a steel-grey color replaces the iridescent red-green and copper bronzing. The gene for narragansett color (*n*) is recessive to N^+ which allows the wild-type bronze to be expressed (Robertson, 1929). Down color of the narragansett poult is phenotypically indistinguishable from wild-type. The grey color is not apparent until six weeks of age. The adult male plumage color consists of light grey feathers with a black edging in the neck, wing fronts, bows, secondary coverts, and the breast. Flight feathers and primary coverts have the usual black and white barring. The feathers on the back are black. The main tail feathers and tail coverts have a broad black band with a white tip. Females have a similar grey plumage with the addition of white edging to the feather tips on the back and breast.

Imperfect albinism. Hutt and Mueller (1942) described a sex-linked recessive form of imperfect albinism derived from bronze turkeys in which nearly all melanic pigment was absent. Day-old albinotic poults had white down, but enough pigment was present to reveal the typical wild-type stripes. These mutant poults were blind; the iris was blue-grey and the pupil was red. Since there was some pigment in the plumage of adult albinos, the feather color was described as dingy-white. In those regions of the feather where black and brown pigment of the bronze pattern would normally be seen, the albinotic turkeys had reduced amounts of pigmentation resulting in a faint barring pattern in the primary flight feathers.

Asmundson (1945a) established that imperfect albinism was actually a recessive allele at the narragansett locus. The symbol for imperfect albinism proposed here is n^{al}. This form of imperfect albinism may be analogous to a similar condition in chickens (Mueller and Hutt, 1941; Hutt and Mueller, 1942).

Brown (*e*). There are two alleles at the sex-linked brown locus. The dominant allele (E^+) permits expression of the bronze plumage color. The mutant allele (*e*) gives brown pigmentation (Asmundson, 1950). The brown phenotype in the day-old poult has the same pattern as the bronze but the down color is reddish-brown. In the adult, the plumage pattern is the same as that of the bronze except that brown pigment is present in place of the typical black and bronze. Barring is still present but it consists of brown and white bars. The brown color is often described as auburn.

Light Brown. When a turkey is genetically both brown (*e*) and narragansett (*n*), a light brown phenotype will result (Asmundson, 1950). The down color of the day-old poult is essentially indistinguishable from brown. But the adult plumage color results in a substantially diluted brown pigmentation resulting in a light brown color, as seen in the Silver Auburn variety.

Faded bronze plumage (*fb*). Keeler et al. (1949) described an autosomal mutant that involved both color dilution and a defect of the nervous system. Down of day-old poults was reddish-brown and it was shortened, most noticeably in the head region. The iris was pink and so were the beak, shanks, toes, and claws. Day-old affected poults were further characterized by abnormal behavior; they exhibited slow movements of the head and poorly directed pecking movements, suggesting a neurological deficit. In older mutants, the bronze pattern had a reddish tinge and feathering was characteristically rough in appearance; many feathers were broken or missing. In adult males, locomotor actions were sluggish and they displayed no mating behavior. Hens had lowered egg production.

Breeding tests suggested an autosomal mode of inheritance. The condition was named faded bronze, but no gene symbol was assigned (Keeler et al., 1949). Somes (1981) provisionally proposed the symbol *fb*.

VARIATIONS IN FEATHERS AND SKIN

Five mutations in turkeys which affect feathers and skin (Table 12.1) have been studied.

Hairy (*ha*). There is only one report of a gene influencing feather structure in the poult. Smyth (1954) described abnormal hairy feathers found in all pterylae of affected birds. Microscopic examination of individual hairy feathers revealed an absence of normal webbing caused by separation of barbs from each other. The major effect of the disorder appeared to be associated with barbule structure and numbers. The hairy condition was detected at hatching and in eggs examined after 25 days of incubation. It was not deleterious to embryo development or to subsequent hatchability, although mutant poults experienced a higher growing mortality. The original hairy poults were derived from the mating of normal individuals and the condition was subsequently established as being an autosomal recessive trait. The mutant gene has been given the symbol *ha*. It may be linked to the gene for red plumage (*r*) but further confirmation is needed.

Late feathering (*K*). The absence of primary flight feathers, or a reduced number of them, in poults at hatching was described as late feathering by Asmundson and Abbott (1961). While the normal day-old poult may have five or six primary flight feathers per wing, the late feathering poult had none or only one or two. By eight weeks of age the late feathering mutant exhibited variation in the degree of feathering from total absence of primaries to a nearly normal condition. At maturity, the late feathering birds could be identified by the absence of flight and tail feathers.

The late feathering condition was inherited as a dominant sex-linked trait and the gene symbol *K* was assigned. A similar mutant is present in chickens (see Chapter 6).

In turkeys, the late feathering gene appears to have deleterious pleiotropic effects on body weight, and shank and keel lengths, but not on reproductive traits. Asmundson and Abbott (1961) advised against commercial use of the late feathering gene because of its pleiotropic effects on body weight. In commercial meat-type chicken production the late feathering gene is used extensively for feather-sexing of day-old chicks. In that species, intense phenotypic selection for body weight in slow feathering birds has overcome the majority of negative pleiotropic or associated effects of the gene. Similar selection might also be possible in the turkey to permit use of the late feathering trait in autosexing of commercial stocks.

Naked (*na*). Poole and Marsden (1961) described a mutation causing nakedness in the Beltsville Small White turkey. Feathering was restricted to the caudal margins of the wings, and to contour feathering on the elbows, wing webs, shoulders and breast. Associated with the naked condition was the presence of one or more extra hind toes on one or both feet, localized to the upper half of the tarsometatarsus. Locomotion was hampered by deformity of the feet and legs. Ataxia was also observed in naked poults.

The condition was inherited as an autosomal recessive trait, and the gene symbol *na* was assigned. Because there were no scales on the feet and no feather follicles on the shanks of affected birds, it was concluded that nakedness in the turkey was not homologous with sex-linked nakedness of chickens (see Chapter 6).

Knobby (*kn*). Smith and Smyth (1970) described a semilethal down abnormality, called knobby, that affected plumule structure in newly hatched poults. Knobby was characterized by large accumulations of undifferentiated cellular material localized in the distal segments of the down plumule. It could be identified by the 17th day of incubation since affected plumules were shortened compared with those from normal embryos. Mortality in the knobby embryos appeared to be a function of the numbers of abnormal plumules present and down coloration. Approximately 60 percent of the affected poults failed to complete development. Although the plumage of juvenile and adult knobby mutants was rough, there were no other apparent feather aberrations observed.

The knobby condition was the result of an autosomal recessive gene with apparently complete penetrance. The gene's expressivity was quite variable suggesting that other loci in the bird's genome may modify its expression. The gene symbol *kn* was proposed by

Smith and Smyth (1970).

Dermal melanosis (*m*). A skin pigmentation disorder in Broad-Breasted Bronze turkeys was described by Harper et al. (1964). Hereditary dermal melanosis was characterized by blue-black skin, which was not related to skin discoloration originating from the melanin of injured feathers. The condition could be detected on visual examination of the crop area in females and the unfeathered surface of the tailhead of both sexes by 12 weeks of age. Melanin deposition visible by 24 weeks of age was sex-influenced; females expressed melanosis in all body surface areas, while male expression was limited to the dorsoventral pelvic girdle. The condition was exacerbated by the bird's plumage color, melanosis occurring earlier in colored birds.

Further studies of the condition revealed that melanotic expression was mediated by exposure to sunlight. Genetically melanotic birds maintained in an artificially lighted facility were compared with siblings maintained under natural sunlight. No dermal melanosis was observed in the confined birds while 89 percent of the birds exposed to sunlight expressed the melanotic condition.

The dermal melanotic condition was the expression of an autosomal recessive gene with the symbol *m*. Although there appeared to be an association of plumage color and melanotic expression, no linkage studies have been conducted.

EMBRYONIC LETHAL DISORDERS

Chondrodystrophy (*ch*). Gaffney (1975) described a chondrodystrophy that was lethal to embryos from a flock of Beltsville Small White turkeys. The mutant embryo was observed to be smaller in size than its normal siblings. Micromelia, brachycephaly, and a pronounced shortening and curving of the lower beak characterized as 'parrot beak' were identifiable after nine days of development. Embryonic death occurred between the 16th day of development and the time of hatch. Occasionally a pipped embryo was observed but none hatched. Typically the long bones were shortened and thickened. The tibia was usually bent in the distal portion of the shaft while abnormal curvature of the femur was also observed. Edema was occasionally observed in the cervical region.

The gene responsible for this form of chondrodystrophy was an autosomal recessive. The designated gene symbol is *ch*. In comparison with Asmundson's (1944) shortened long bones mutation (*s*), heterozygous Ch^{+}/ch birds did not differ from normal wild-type.

Chondrodystrophy-m (*ch-m*). Another type of chondrodystrophy in embryonic turkey poults was observed by Nestor (1978) in an inbred line of Large White turkeys. The affected poults were characterized by shortened and thickened long bones of the legs and wings, and proportionally shortened beaks. Terminal phalanges were missing in about half of the mutants. A hydrocephalic condition was also observed in some affected embryos. It differed from that described by Gaffney (1975) in that parrot beak was not observed. The condition was regarded as semilethal and mutants reared to maturity never developed sexually. As with many embryonic semilethal disorders, the affected poults required assistance to hatch.

This form of chondrodystrophy was the expression of an autosomal recessive gene. The gene symbol ch^{m} was assigned by Nestor (1978), but the symbol was subsequently changed to *ch-m* by Somes (1981).

Crooked neck dwarf (*cn*). A lethal embryonic condition similar to the crooked neck dwarf mutant in chickens (Asmundson, 1945b) was observed in the turkey by Asmundson and Pun (1956). It was characterized by reduced body size, a slightly curved and shortened upper beak, rigid cervical vertebrae with concomitant head turning to either the right or left, muscular hypoplasia in the tibiotarsal section, rigid tibiotarsometatarsal joints, and curled phalanges. Although poults were alive on the 26th day of incubation or later, they did not pip the shell. Hydramnios was present with the fluid usually clear or having a greenish tint. Histological examination revealed the presence of esophageal atresia. Organ weights of the lethal embryos were reduced as described in the chicken homologue, with greatest reductions in the thyroid and testes. Observations indicate that recessive white plumage (*c*/*c*) does not influence the mutant phenotype (Savage, unpublished). This embryonic condition has also been observed in Japanese quail embryos (Sittmann and Craig, 1967) but with morphological differences; the phalanges were spread apart, body size was reduced although not as acutely as in affected chickens and turkeys, and there was no esophageal atresia.

The lethal condition in the turkey is the expression of an autosomal recessive gene with minimal variation in expressivity. The gene symbol *cn* used to identify this gene is the same as that used in chickens and Japanese quail.

Hemimelia (*hm*). An embryonic condition characterized by the absence of distal parts of the hind limbs was described by Abbott and Asmundson (1962). The hemimelic embryos had normal wings; tibia and fibula were normal, but the more distal leg bones in one or both legs were absent. Other variations observed consisted of reductions in the tarsometatarsal bones and their associated digits and occasional juxtaposition of the digits to the proximal end of the hock joint. The mutant embryos were smaller than their normal siblings and may also have had reduced maxillae and celosomia.

The disorder was inherited as an autosomal recessive trait with variable expressivity. The gene symbol *hm* was proposed. The *hm* gene expression appears to be associated with a defective development of the apical ectodermal ridge.

Micromelia-like (*mm*). Cramer and Harper (1975) described a lethal micromelia in midget-type turkeys that resulted in the inability of the poult to pip the egg. The micromelic embryos had shortened legs, a curved premaxilla, reduced lower mandible, and an overall shortening of the appendicular bones. Measurements of the tarsometatarsus revealed a 57 percent reduction in bone length, but the thickness was increased by 148 percent. Another prominent feature of this mutation was the entanglement of the yolk sac membrane around the left leg above the digits, suggesting that development of a body malposition was characteristic of this micromelic condition.

Selected matings suggested that the condition was due to an autosomal recessive gene. Somes (1988) assigned the gene symbol *mm* (micromelic-like). This micromelia condition has some similarities to Asmundson's (1944) short long bones mutant where the long bones were also reduced, but they differed in structure of the mandible. The short long bones mutant was described as being semilethal whereas micromelia-like is lethal.

Ring lethal (*rl*). Ring lethal (Savage and Harper, 1985) is an embryonic failure occurring within the first 48 h of incubation. The condition was characterized by an amorphous mass of cells in the *area opaca* with or without the presence of cells in the *area pellucida*. The outer diameter of the ring structure ranged from 4-12 mm, and the width of the ring measured 1.6 mm. Variability in expression of the ring disorder ranged from a mass of white cells to a structure with two concentric white rings. Blood islands were observed in only 0.7 percent of affected embryos. Parthenogenesis could be mistaken for the ring lethal because both can have a ring-like structure, but the diameter of unorganized membranes in parthenogenesis is usually greater than that of the ring lethal.

The condition was inherited as a single autosomal recessive gene and the symbol *rl* was proposed for it (Savage and Harper, 1985). Although the precise mechanism for *rl* gene expression remains unknown, it may be associated with the synthesis of a defective protein that permits adhesion between the dividing embryonic cells and the vitelline membrane.

Shortened long bones (*s*). A semilethal skeletal abnormality characterized by shortening of the long bones (femur, tarsometatarsus, humerus and radius) was described by Asmundson (1944) in Bronze turkeys. This shortened long bone condition affected the viability of the developing embryo by preventing emergence from the shell; only 19 percent of the mutants hatched. Those poults with shortened bones that were able to hatch exhibited reduced viability because of their inability to compete for food and water.

This condition was inherited as an autosomal trait, and it has been classified as recessive with the gene symbol *s*. However, it may actually be incompletely dominant, since heterozygotes have slightly reduced long bones.

Short-spined (*sh*). A lethal embryonic disorder, in which poults failed to pip the shell and had shortened body lengths, was described as short-spined by Asmundson (1942). He observed this mutant in a line of inbred Bourbon Red turkeys. The mutant was characterized by compaction of the space between the cervical vertebrae, resulting in crowding of the neck vertebrae and reduced body length. The lengths of wing and leg bones were not affected. Expressivity of the short-spined condition ranged from embryos with normal-sized skulls to others with size reduction in skull, eyes, and upper beak. The ash percentage was less in the scapula, ilium, and ischium of short-spined embryos when compared with normals. Lethality appeared to be associated with inability of the poult to pip the shell. The short-spined condition differs from other known skeletal disorders of turkeys since only the axial skeleton is affected by the mutation.

The short-spined condition was due to an autosomal recessive gene. Although a gene symbol was not proposed when the mutation was initially reported, Somes (1981) has suggested the symbol *sh*.

Swollen down plumules (*sdp*). Swelling of the proximal segment of the down plumules in turkey embryos was reported by Savage et al. (1986). The condition has been found in commercial strains of Large White turkeys and in a strain of Wrolstad Medium Whites. It has been detected as early as the 20th day of incubation. Affected poults usually die prior to pipping. Those assisted from the egg do not survive longer than 3-4 days. Severity of expression varies from poults with swollen plumules localized on the ventral neck surface to others in which all the pterylae of the embryo are occupied with abnormal plumules. Gross necropsy of mutant embryos has not revealed any abnormality of the internal organs. Histological examination of the swollen plumules revealed a distended dermal pulp cavity containing large quantities of disorganized pulp material.

Swollen down plumules is the expression of an autosomal recessive gene (*sdp*) with variable expressivity. Although the mechanism for gene expression remains undetermined, chicken phenocopies of the condition can be produced by treatment of developing embryos with retinoic acid (Dhouailly and Hardy, 1978). Histologically, the abnormality induced by retinoic acid resembles that of the turkey mutant.

Another embryonic feather plumule abnormality that has some resemblance to the swollen down plumules mutant is that of clubbed down (Hawes and Buss, 1963). The swelling of clubbed down plumules is usually in midshaft and it is made worse by the presence of the black plumage allele. Typically, clubbed down is associated with a dietary riboflavin deficiency (Lepkovsky et al., 1938). Hawes and Fox (1962) reported amelioration of the clubbed down syndrome by means of riboflavin supplementation. However, embryos with swollen down plumules do not respond to riboflavin treatment (Savage, unpublished).

NEUROLOGICAL MUTATIONS

Mutations whose phenotypes have neurological expression in turkeys are very few. Of the three that have been reported, two are expressed at hatching and the third manifests itself later.

Congenital loco (*lo*). Knowlton (1929) is credited with the first description of congenital loco; he found it in chickens. Its presence in the turkey was reported by Cole (1957), where it is considered to be an obligate lethal since affected poults usually die of starvation or dehydration within a few days after hatching. The loco poult is characterized by continuous retraction of the head (opistotonic spasms) with occasional lateral head and neck movements. Often affected poults simply lie on their sides. The morphological basis for mutant expression is undetermined although Bernier (1988, personal communication) has observed day-old poults with a similar condition to have fused otoconia in the inner ears.

Congenital loco in the turkey is the expression of an autosomal recessive gene with the gene symbol *lo* (Cole, 1957). Birds heterozygous for this disorder appear normal and are reproductively viable. Similar conditions are known in chickens and Japanese quail, and they may be genetically homologous (Knowlton, 1929; Sittmann et al., 1965).

Vibrator (*vi*). The second inherited neurological disorder to be reported for turkeys was vibrator (Coleman et al., 1960). This mutation, observed in a strain of Broad-Breasted Bronze, was expressed at hatching time as a rapid shaking or vibration of the head and neck. Although the condition was not lethal at hatching time, Kulenkamp et al. (1968) reported that there was a significantly higher incidence of mortality between hatching and eight weeks of age when compared with normal sibs. The vibrator expression diminishes with age, and it can be difficult to identify in adult birds. There was no apparent influence of the vibrator disorder on female reproductive performance; the effect of the disorder on reproduction in males has not been studied. Attempts at identifying a neuro-anatomical anomaly associated with the vibrator condition were unsuccessful (Peterson et al., 1963).

The gene responsible for the vibrator condition is sex-linked recessive with the gene symbol *vi* (Coleman et al., 1960).

Bobber (*bo*). The third neurological mutation to be studied and reported in the turkey is bobber (Harper et al., 1988). This condition expressed itself between 14 and 21 days of age by cervical bending of the neck in an anterior direction so that the head became positioned between the legs. This mutant, when in a nonexcited state, is capable of raising its head to a nearly normal position to eat and drink. When subjected to a loud noise the bobber resumes its abnormal head posture. Affected birds have also been observed to move rapidly in a counterclockwise pattern described as pirouetting. The bobber when suspended by its feet will demonstrate an atypical orientation of the head and neck, drawing its head toward the breast. Once expressed, the bobber condition remains with the bird throughout its life. The mutant poults have a high incidence of mortality, associated with inability to locate feed and water. Reproductive capability is not impaired in either sex.

Expression of the bobber gene has been associated with an age-influenced diminished synthesis of plasma norepinephrine (Harper et al., 1988). A transient amelioration of the bobber posture is achieved by intravenous injection of norepinephrine. A permanent correction of this condition is achieved by exposure of the bobber to intense sunlight. This finding represents a second case (see dermal melanosis) where sunlight has a definitive effect on expression of a mutant gene; the dermal melanosis (*m*) mutant is also responsive to sunlight.

Bobber has been shown to be a sex-linked recessive trait (Harper et al., 1988). The gene symbol *bo* has been proposed for it.

METABOLIC MUTATIONS

Binucleated erythrocytes (*bn*). Buss (1965) reported a hematological abnormality where circulating red blood cells contained nuclei of two unequal sizes. They were described as binucleated red blood cells (BRBC). The condition was detected in bone marrow cells of poults up to two days of age, and about a day later they could be found in the circulating blood supply. The incidence of BRBC reached its maximum at about six weeks of age. Females had a higher incidence of the abnormality than did males. Average incidence in adults was five percent, but by selection Bloom (1977) raised it to 40 percent in a subline and to 60 percent or more in some individuals.

The BRBC condition was described as resulting from an autosomal recessive gene with the symbol *bn* (Buss, 1965). However, Bloom et al. (1970) noted in subsequent studies that a few BRBCs occurred in heterozygotes (Bn^+/bn). This raises the question of whether the mode of inheritance is really recessive. Studies of mutant gene expression (Searle and Bloom, 1979) suggest that the *bn* gene affects the structure or function of the cell's spindle apparatus during metaphase and anaphase stages of cell division.

Bowed hocks (*bh*). The expression of leg weaknesses in birds is sex-influenced, with males having a higher incidence than females. Buss (1978) briefly reported that one cause of leg weakness was a condition referred to as bowed hocks. He determined that bowed hocks in turkeys at 24 weeks of age was the result of an autosomal recessive gene. Somes (1988) proposed the symbol *bh* for this gene.

Hereditary glaucoma (*ga*). An ocular glaucoma condition characterized by corneal protrusion and increased curvature, expressed between the time of hatching and 22 weeks of age, was described by Smyth et al. (1982). The mutation was observed in a line of turkeys homozygous for the dominant slate plumage mutant. Variable changes in intra-ocular pressure occurred as the glaucoma condition developed, followed by decreased pressure as the condition progressed. Histological examination revealed corneal edema, and enlargement of the anterior chamber in early stages of disease development, with varying degrees of corneal opacity and buphthalmus.

The condition is due to an autosomal recessive gene with incomplete penetrance (Smyth et al., 1982). The provisional gene symbol *ga* has been proposed (Somes, 1988).

Muscular dystrophy (*dy*). A dystrophic condition affecting the breast muscles of young growing Broad-Breasted Bronze turkeys was described by Harper and Parker (1967). Atrophy of the *pectoralis major* and *pectoralis minor* was visually apparent after eight weeks of age, resulting in approximately 40-60 percent reduction in muscle mass. This was accompanied by degeneration of the alar wing muscles. Between 32 and 36 weeks of age, atrophy of the pectoralis muscles in afflicted turkeys was complete. A unique characteristic of the syndrome was the inability of the bird to right itself when placed in a supine position. Also noted was an unusual increase in the muscle mass of the legs of dystrophic females. Unlike the situation in muscular dystrophy of chickens (Asmundson and Julian, 1956), muscle hypertrophy did not precede atrophy in the mutant turkey. Microscopic examination of dystrophic turkey muscle fibres revealed an infiltration of adipose cells into the space previously occupied by muscle tissue. Histopathological studies indicated that the defects were of myogenic origin (Schmitz and Harper, 1975).

The gene responsible for turkey muscular dystrophy is autosomal recessive, with minimal expressivity and complete penetrance (Harper and Parker, 1967). The symbol *dy* was proposed.

Pendulous crop. An abnormality of the ingluvies or crop characterized by distention due to excessive liquid consumption was described by Asmundson and Hinshaw (1938). The condition, expressed as early as eight weeks of age, was capable of remission followed by re-expression. From mating results, the condition was determined to be caused by an autosomal recessive gene with incomplete penetrance. Phenotypic expression was influenced by environmental temperature and humidity, and by plumage color.

No gene symbol was suggested since there was concern that a modifying gene at another locus was also involved (Asmundson and Hinshaw, 1938). Somes (1981) has interpreted the inheritance of pendulous crop to be polygenic.

BIOCHEMICAL VARIANTS

Studies of protein variation in turkeys are few in number, with little interpretation available regarding significance of the variation. The commonly used method for identifying biochemical variation has been starch-gel electrophoresis, because of its ease of application, but at the expense of protein resolving potential. There is in this author's opinion considerable biochemical variation present in the turkey. Investigative research needs to be conducted. Biochemical variation, whether it is qualitative or quantitative, must have a physiological and underlying genetic basis for its presence. Genetic variation is not sustained without biological justification. Refer to Chapter 8 for a general discussion on biochemical variation.

Albumin. Polymorphic blood albumins were observed by Quinteros et al. (1964) following electrophoresis of plasma from progeny of the interspecific cross between domestic (*M. gallopavo*) and ocellated turkeys (*M. ocellata*). The three phenotypes identified were designated as A, B, and AB. Phenotypes A and B represented homozygotes and each contained two protein bands. The fastest migrating band of the two in phenotype A corresponded with the slowest migrating band in phenotype B. The heterozygote AB exhibited the three electrophoretic bands observed in both homozygotes. The genes occupying the albumin locus are autosomal and co-dominant. The gene symbols are Alb^A and Alb^B.

Adenosine deaminase. Grunder and Hollands (1978) described a red blood cell lysate polymorphism of adenosine deaminase (EC 3.5.4.4) in a strain of commercial Small White turkeys. Three phenotypes were observed in the samples. They consisted of two different single zone patterns of enzyme activity of varying anodic electrophoretic mobility, designated A and B, or a pattern with both zones which was designated AB. Examination of other tissues from the birds studied revealed a phenotypic homology with variation in the blood lysates. Expression of the zones was controlled by two autosomal co-dominant alleles designated as *Ada-A* and *Ada-B*.

Alkaline phosphatase. Variation in the presence of plasma alkaline phosphatase (EC 3.1.3.1) isoenzymes has been described by Stevens and Garza (1968). Of four isoenzymes designated Akp-1 through -4, either Akp-2 or Akp-3 was consistently present. *Akp-2* was determined to be autosomal and completely dominant to *Akp-3*.

Lactate dehydrogenase. Polymorphism for a turkey lactate dehydrogenase (EC 1.1.1.27) was briefly described by Kutnyuk (1978) using starch-gel electrophoresis. Two autosomal co-dominant genes designated as *Ldh-S* and *Ldh-F* resulted in three electrophoretic phenotypes. The tissue source for the enzyme was not identified.

IMMUNOLOGICAL VARIATION

Qualitative immunological variation in the turkey, as with the topic of biochemical variation, has received minimal research attention. Seven different blood group antigenic systems have been identified in the turkey (Stevens and Asmundson, 1967). Three of the antigenic groups, designated A, C, and F, have multiple alleles. Somes (1981) proposed using the erythrocyte antigen gene symbol *Ea-* in combination with the appropriate blood group letter and number designation to identify genes in the systems.

Palmer and Nordskog (1980) identified two major histocompatibility alleles in the turkey, but they were unable to associate these with erythrocyte antigens.

Refer to Chapter 22 for a full discussion on immunogenetics, particularly as pertaining to chickens.

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Chapter 13. MUTATIONS AND MAJOR VARIANTS IN JAPANESE QUAIL

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The Japanese quail (*Coturnix japonica*) is a close relative of the chicken and the pheasant. It was domesticated in the 11th century, and was used first as a song bird and then as a meat and egg producer; it later became popular as a laboratory animal (see Chapter 1). Because of its small body size, it has never been as popular as the chicken for meat production, and has remained in the specialty food category. Although it has been widely consumed in the Middle East, Asia and Europe, its consumption in North America and Australia has been low until only very recently.

The number of reported mutations and major variants in Japanese quail is small compared to those reported in the chicken, and not studied as closely. This is true for several reasons. Although the amount of research involving Japanese quail has been large, in the majority of these studies the bird has been used as a model or for comparison purposes, and few researchers have been interested in the bird itself. Because of its small body size, many morphological changes can escape detection unless the bird is handled regularly and close attention is paid to it. During selection programs, poultry geneticists may pay attention to morphological changes in chickens in search of easily identifiable markers for less easily measured physiological traits. The amount of selection in Japanese quail has been minimal, and there has not been much need for morphological markers. Many recessive mutations in chickens became homozygous and noticeable in the process of developing inbred lines. Japanese quail are much more susceptible to inbreeding depression, and with only a few exceptions, attempts to form inbred lines have not been very successful. In chickens, pigeons, and waterfowl species, many color variants were first observed and maintained by fanciers and hobbyists, and were later made available for analyses. Because of its cryptic coloration and behavior, the Japanese quail is not as common as these other species in aviaries, especially since its popularity as a song bird in Japan subsided after World War II.

In this chapter, an attempt will be made to summarize and discuss mutations found in Japanese quail with reference to those found in other domestic species, especially the chicken, and to point out where future research may be necessary. The gene symbol conventions proposed for poultry by Somes (1980) will be followed. In some cases, the gene symbol used in the original reports has been changed to conform to these conventions. Whenever possible, the original gene symbol will be mentioned and the reader will be referred to the original reports.

GENETICS OF PLUMAGE COLOR AND PATTERN

The natal plumage of wild-type Japanese quail chicks is similar to that of other galliforms but it is more dusky. The head is tawny with a small black patch above the beak. There is a median buff stripe along the top of the head bordered by black stripes. The back and the wings are light brown and there are four heavy blackish brown stripes on the back.

The juvenile plumage is completely grown at about four weeks after hatching. This plumage is very similar to the adult female plumage except that the cream median streaks on the back feathers are narrower, and the markings on the face are not as distinct. Juvenile distal primaries have sharp tips whereas adult primaries have blunt tips.

The plumage of wild-type adult Japanese quail is brown in color (Figure 13.1), but it is highly variable in terms of the shades of brown and some of the markings on the breast and throat. In adult males the forehead, crown, and nape are black with rusty brown edges. The median and superciliary lines are creamy white. The back contour feathers are greyish black with broad cream shaft streaks and thin cream transversal bars; the greyish black turns into a rusty brown color towards the tip of the feather. The wing coverts are greyish brown; the shafts are grey turning to cream color at the tips; the coverts are transversed with five or six bars of cream color sandwiched between dark brown. The throat is reddish brown and the breast is orange-brown. The abdomen and under-tail coverts are pale cream in color. Adult females are similar to males in color, except that the throat and breast are creamy white with dark brown dots (see also Wetherbee, 1961; Taka-Tsukasa, 1935).

Under a short light cycle, the adults may develop a much paler winter plumage; the feathers on the chin and the throat lengthen, become white, and more pointed at the tip. Male and female plumage becomes more similar.

The genetics of plumage color in chickens has been reviewed in Chapter 5. The melanins (eumelanin, pheomelanin, and the pheomelanin-related trichochromes) are the pigments that determine the plumage color and patterns of chickens. It is the intricate interaction of genes that affect the presence and distribution of melanins with genes or gene complexes that affect feather structure which give rise to the seemingly infinite variation in plumage color found in domestic fowl. Although the number of reported color variants in Japanese quail is very limited by comparison, the same genetic mechanisms should apply. So far most of the plumage color mutations found in Japanese quail have been merely documented and described. With the exception of studies by Somes (1976, 1979) and Truax and Johnson (1979), little effort has been made to unravel intra- and interallelic interactions. With recent findings that male Japanese quail with different throat color patterns show differences in photo-induced testicular growth and regression (Oishi and Konoshi, 1983; Burke and Siopes, 1988), and that several color variants in Japanese quail show genetically different ocular response to light stimulation (Lauber and Cheng, 1989), more intensive studies of color mutations in Japanese quail are certainly justifiable.

In this section, the various plumage color mutations are reviewed by groups: first, those mutations that extend the distribution of melanin; secondly, those that dilute pigmentation; thirdly, those that restrict distribution of melanin; and finally, the amelanotics (Table 13.1). However, there are some mutations that fall into more than one group.

Extension Of Melanin Pigmentation

As reviewed in Chapter 5, the distribution of melanin can be classified into primary patterns (regional, involving several feather tracts) and secondary patterns (within individual feathers). Eumelanin gives rise to black while pheomelanic pigments give rise to red and buff colors. There are three reported mutations involving the extension of pigmentation in Japanese quail: extended brown, black-at-hatch, and dark feather nervous disorder. The latter will be discussed under Neurological Mutants.

Extended brown (*E*), black (*D*). The extended brown (*E*) gene is carried in British

Table 13.1. Japanese quail plumage color mutations and their gene symbols.

Trait	Gene symbol	Reference
Extended brown	E, e^+	Somes (1979)
Black-at-hatch	Bh, bh^+	Minezawa and Wakasugi (1977)
Dark feather nervous disorder	dn, Dn^+	Kawahara (1979)
Red-head	e^{rh}, e^+	Truax et al. (1979)
Pansy	ps, Ps^+	Tsudzuki and Wakasugi (1987)
Yellow	Y, y^+	Homma et al. (1967)
Fawn	Y^F, y^+	Nichols and Cheng (1988)
Cinnamon	cin, Cin^+	Fulton et al. (1982a)
Buff	pk, Pk^+	Sittmann et al. (1966a)
Dark-eyed dilute	al^D, Al^+	Chahil and Johnson (1974)
Sex-linked brown	br, Br^+	Homma (1968)
Roux	ro, Ro^+	Perramon (unpublished)
Dominant white	W, w^+	Wakasugi and Kondo (1973)
Bleu	bl, Bl^+	Perramon (unpublished)
Recessive silver	rs, Rs^+	Watanabe and Homma (1982)
Marbled plumage	ma, Ma^+	Yakovlev et al. (1975)
Recessive white	wh, Wh^+	Roberts et al. (1978)
Panda	$wh, Wh^+ (s, S^+)$	Mizutani et al. (1974)
Brown-splashed white	p, P^+	Shimakura (1940)
Silver feathered	B, b^+	Homma et al. (1969)
White-breasted	wb, Wb^+	Roberts et al. (1978)
White crescent	cr, Cr^+	Roberts et al. (1978)
White bib	bi, Bi^+	Roberts et al. (1978)
White primaries	wp, Wp^+	Somes (1984)
White-feathered down	c, C^+	Sittmann and Abplanalp (1965a)
Complete albinism	a, A^+	Homma et al. (1968)
Imperfect albinism	al, Al^+	Lauber (1964)

Range, English White, and Tuxedo strains (Somes, 1979). Extended brown is incompletely dominant to wild-type (e^+). Homozygous brown individuals are uniformly dark brown in both the ventral and the dorsal plumage, with a small area of white feathers around the beak and on the throat under the lower beak (Figure 13.1). The extension of melanin mainly affects the secondary pattern. Heterozygous (E/e^+) individuals exhibit the shafted phenotype. Their dorsal feathers have the dark striped pattern similar to that of the wild-type. The rest of the feathers are brown with some having dark tips. The pattern on the feathers is similar to that of the wild-type except that it has a darker brown than in wild-type coloration. The shafts of the body feathers are tan instead of cream-colored. Homozygous brown chicks have black down ending with brown tips on the head and on the back. The face and throat is creamy yellow and the ventral surface is a mixture of grey and creamy-yellow. Heterozygous chicks have brown down on the back and on the head and show the three longitudinal dark stripes on their back similar to the wild-type. Extended brown is epistatic to yellow (*Y*) and hypostatic to recessive white, and it segregates independently of white-breasted (*wb*), rough-textured (*rt*), short-barb (*sb*), and ruffle (*rf*) (Cheng, unpublished), as well as from hemoglobin (*Hb-1*), and transferrin (*Tf*) (Ito et al., 1988a). Extended brown is linked to phosphoglucose isomerase (*Pgi*) with a recombination rate of about 31 percent for the males (Ito et al., 1988a), and about 43 percent for the females, an interesting sex difference. While the gene symbol *E* is used, this locus in

Japanese quail may not be homologous to the *E* locus in chickens. The multiallelic *E* locus in chickens can be considered as the locus determining the basic distribution of eumelanin (see Chapter 5). In Japanese quail, only one other allele (e^{rh}) besides the wild-type (e^{+}) has been found for the *E* locus. Only hybridization experiments will determine whether there is homology between species.

Chikamune and Kanai (1978) reported a dark mutation which was later called black by Somes (1984) and given the gene symbol *D*. Based on description of the phenotype and its epistatic relationship with recessive white (*wh*) (Chikamune and Kanai, 1978), black is likely to be homologous with extended brown.

Black-at-hatch (*Bh*). The *Bh* trait is controlled by an autosomal dominant gene with homozygous lethality. Homozygous (*Bh*/*Bh*) embryos die between 4.5 and 8 days of incubation (Minezawa and Wakasugi, 1977; Ono and Wakasugi, 1983b). The primary defect is suspected to be the disintegration or dysfunction of capillaries, causing deficiency in the oxygen supply, subsequent degeneration of the left lobe of the liver, and internal hemorrhage (Ono and Wakasugi, 1983a). Heterozygous chicks lack the yellow dorsal markings of wild-type chicks. Microscopic observations reveal that the dorsal down of wild-type chicks has both pure black and pure yellow barbules. The down of heterozygous chicks has barbules that are either pure black or a mixture of black and yellow pigments, but none are pure yellow. Difference in the plumage of heterozygous and wild-type adults is less conspicuous. Most of the back contour and wing feathers of the mutant lack the cream-colored transversal bars of the wild-type but show intermediate coloration between black and yellow. The *Bh* locus segregates independently and shows no linkage with yellow, white (*W*), silver (*B*), and white egg-shell (*we*) (Minezawa and Wakasugi, 1977), nor is there linkage with *Hb-1*, *Pgi*, and *Tf* (Ito et al., 1988a).

Dilution Of Color

The dilution of color can involve both the dilution of pigments and a restricted distribution of pigments affecting primary and/or secondary patterns. The majority of color variants in Japanese quail fall into this category (Table 13.1).

Redhead (e^{rh}). A mutant called redhead was reported by Truax et al. (1979) and was found to be allelic to extended brown. Redhead (e^{rh}) is recessive to *E* and e^{+}. The adult plumage of the mutant is predominantly white with irregular black and rust markings (Figure 13.1). The underfluff of the feathers is smokey grey. The breast feathers of the females are white with a rust tinge, and with a black bar towards the tip. The shaft color varies from black in the upper breast feathers to white in the lower breast feathers. The breast feathers of the males are a mixture of white and rust without any black bars. The top of the female head has a black cap that extends down the back of the neck. The males' head is a bright rust color. The chicks have light yellow down with a reddish tinge on the head, and the longitudinal stripes show faintly on the back. In the homozygous state, redhead is epistatic to recessive white and yellow.

Pansy (*ps*). Tsudzuki and Wakasugi (1987) found an autosomal recessive mutation with a phenotype almost identical to that of the redhead both as chicks and as adults. The authors concluded that this mutation was not homologous to the redhead after comparing subtle differences in the feather pattern of the two birds. The mutation was called pansy (*ps*). However, there is some variation in shade of plumage color and in amount of markings in populations of redheads. The redhead feathers available to Tsudzuki and Wakasugi for comparison may not have represented the whole spectrum of variation. By comparing feather samples of pansy sent by Dr. Wakasugi with feathers from the redhead population at the Quail Genetic Stock Centre, University of British Columbia, it can be concluded that pansy certainly falls within the variation of redhead. Since redhead is not available in Japan, an allelic test between pansy and dark (presumably the same as extended brown) is being carried out; preliminary data indicate that pansy is allelic to dark (Wakasugi, 1988, personal communication), and therefore it is very likely that redhead and pansy are homologous.

Fawn (Y^F). Another mutation with phenotype similar to the redhead was described by Somes (1984). The male mutant has a light buff to fawn coloration. This is mainly because the cream color of the shaft streak and the transversal bars of the back and contour feathers has been extended to the whole feather. What is left of the dark pigmentation is just a tinge of brown around the edges of the feathers. The females have some spots of darker color on the dorsal and breast plumage; in these areas, the feathers still retain a streak of dark pigment along the edges. The head of the male is a dark rust color. The mutation was called fawn but the mode of inheritance was not determined until recently (Nichols and Cheng, 1988). Fawn (F) is incompletely dominant to y^+ and codominant to yellow (Cheng, unpublished), but the homozygotes are not lethal. In the heterozygous state, the back feathers of both males and females retain a dark pigment streak along the edges, and the streaks are thicker than those on the back feathers of homozygous females. This gene is carried in commercial strains called Australian Fawn or Speckled Fawn.

Yellow (Y). The Yellow variant, an autosomal dominant mutation, was found by Homma et al. (1967). The gene is carried in a commercial strain called Manchurian Golden. Homozygotes (Y/Y) are lethal, with mortality occurring before 48 h of incubation. Roberts and Fulton (1980) suggested that heterozygotes (Y/y^+) may also suffer ten percent mortality (see also Kraszewska-Domanska and Pawluczuk, 1977). In the heterozygous state, the mutant is a golden wheat-straw color. The general pigment distribution is the same as in the wild-type except that the cream-colored shafting of the back and hackle feathers is much wider and the wing-bow and head feathers are also wheat-straw yellow color. Yellow (Y/y^+) chicks show the dark longitudinal stripes of the wild-type but the rest of the down is of a rich cream color. Yellow in the heterozygous state is hypostatic to homozygous extended brown and redhead, but (Y/y^+ E/e^+) adults express a yellow-shafted phenotype (Truax and Johnson, 1979), and chicks have yellow-tipped down on the head (Roberts and Fulton, 1980). Yellow is also epistatic to recessive white (Chikamune and Kanai, 1979), and segregates independently of recessive white, extended brown, short-barb (*sb*), and rough-textured (*rt*) (Cheng, unpublished), and independently of *Hb-1*, *Pgi*, and *Tf* (Ito et al., 1988a). Yellow is linked to white-breasted (*wb*) with a distance of 36.9 map units (Cheng, unpublished).

In comparing the back feathers of Y, e^{rh}, and Y^F, the differences are both obvious and interesting. In the yellow, both the cream-colored shaft streak and the transversal bars are widened, but the pattern is still distinct. In the redhead, the transversal bars are widened but not the shaft streak, and the pattern becomes diffused resulting in the appearance of two dark and difussed bands on a creamy buff feather. In the heterozygous fawn, the shaft streak is widened more so than the transversal bars resulting in what seems to be a streak of dark pigment along both edges of the feather. In the homozygous fawn, all of the dark pigment has disappeared from the feather. The degree of dilution seems to be least in the yellow, most in the fawn, with the redhead in between. This difference in the degree of dilution is also reflected in the amount of pigmentation of their primaries and in the natal plumage of their chicks. The modes of inheritance, however, are very different for the two loci.

Cinnamon (*cin*). Cinnamon is an autosomal recessive mutation found by Fulton et al. (1982a) in a population homozygous for extended brown. The mutation is a dilution of the dark brown pigments of *E*/*E* to a cinnamon color. Individuals with genotype $cin/cin\ e^+/e^+$ express a phenotype that is very similar to the autosomal dilute (W/w^+) (Cheng, unpublished). Recently Takahashi et al. (1988) reported that a spontaneous recessive mutation affecting plumage color was detected in their strain of Japanese quail selected for 22 generations for low titre to inactivated Newcastle disease virus vaccine. The phenotype of this mutant is very similar to that of *cin*/*cin* (Cheng, unpublished). This mutation has been fixed and used as a marker for the low titre strain.

Buff (*pk*). Another mutation that dilutes plumage color but does not affect plumage pattern is buff (Sittmann et al., 1966a). Pigmentation of the eye is also affected by this autosomal recessive mutation and the mutant phenotype resembles very closely the pinkeye mutation in chickens. Sittmann et al. (1966a) believed that the two mutations were homologous and adopted the gene symbol *pk* assigned for the gene in chickens. Compared to imperfect albinos, buffs have a higher although still subnormal intensity of melanin in the retina and the choroid. Melanin is absent in the ciliary body and the inner layer of the iris of buffs but found in reduced intensity in imperfect albinos. Hatchability, posthatch viability, and fertility of the buffs are markedly reduced compared to wild-type birds. Buff is epistatic to imperfect albinism.

Dark-eyed dilute (al^D), sex-linked cinnamon (al^c), red-eyed brown (*reb*). Dark-eyed dilute was reported by Chahil and Johnson (1974) and found to be an allele at the sex-linked *al* locus. Dark-eyed dilute is recessive to wild-type but dominant to imperfect albino. Although it is called dark-eyed dilute, the eyes of the chicks are really dull red and have subnormal melanin pigmentation. The eyes darken with age but in some adults there is still a reddish tinge when examined closely. Homozygous (al^D/al^D) and al^D/al individuals have dilution of the brown pigments of the feathers but the wild-type plumage pattern is not affected. Wakasugi and Kondo (1973) also reported a sex-linked recessive mutation which is allelic to imperfect albinism. They named the mutant red-eyed brown (*reb*) but later Mizutani referred to the same mutant as sex-linked cinnamon (al^c) (Truax and Johnson, 1979). An examination of back and wing feathers of sex-linked cinnamon shows that they are indistinguishable from those of the dark-eyed dilute (Cheng, unpublished) and the two mutations seem to be homologous.

Sex-linked brown (*br*), roux (*ro*). Homma (1968) reported a sex-linked recessive mutation called sex-linked dilute. Wakasugi and Kondo (1973) referred to the same mutation as sex-linked brown. Adult female mutants resemble the dark-eyed dilute except the shaft and white parts of the feathers are a reddish sandy color in sex-linked brown. Adult homozygous (*br*/*br*) males are also reddish diluted brown but are darker than the females. The wild-type plumage pattern in either sex is not affected by the mutation. Wakasugi and Kondo (1973) found a 35 percent recombination rate between *br* and *al*. Perramon (Somes, 1984, personal communication) found a sex-linked recessive color mutation called roux (*ro*) and found a 30 percent recombination rate between *ro* and *al*. Roux and sex-linked brown have similar phenotypes and may be homologous, but unfortunately no testcross has been made.

White (W), autosomal dilute, dominant dilute (*Dil*). A white mutant (W), reported by Wakasugi and Kondo (1973), is incompletely dominant. This mutation is reviewed here because the heterozygotes (W/w^+) have a diluted coloration. The homozygote (W/W) is completely white-feathered with black skin. It is semilethal, very few individuals surviving to adulthood. The W gene is therefore maintained in populations in the heterozygous state. The heterozygotes were mistakenly identified as a different mutation called autosomal dilute by Somes (1984) and dominant dilute (*Dil*) by Truax and Johnson (1979), probably because of errors in translation (Mizutani, 1988, personal communication). White segregates independently from *Hb-1*, *Pgi*, and *Tf* (Ito et al., 1988a).

Marbled plumage (*ma*). In three plumage color dilution variants, pheomelanin is affected more so than eumelanin, resulting in grey, blue, and silver colors. After exposing a group of quail to x-irradiation, Yakovlev et al. (1975) discovered two mutant chicks among a group of 5000 progeny. The chicks had light blue to white down with normal eye pigmentation. As adults, the birds had "light-blue to white plumage with translucent veins (vanes)". The trait was named "marbled plumage" (*ma*). Presumably it was induced by irradiation. In the initial generations there was high frequency of embryonic mortality associated with the trait, but in the later generations the authors were able to select for marbled plumage birds with low embryonic mortality, and a line of these birds has been established.

Bleu (*bl*). This recessive mutation replaces the brown color of the wild-type with bluish grey and the cream-colored markings with white (Perramon, 1988, personal communication).

Recessive silver (*rs*). This autosomal recessive color mutant was first reported by Watanabe and Homma (1982). Egg production of homozygous females is affected; Homma et al. (1985) found that although homozygous females are able to ovulate, they are unable to secrete albumen. This is in spite of immunophoretic and histochemical evidence that their oviductal tissue contains conspicuous amounts of albumen. Watanabe and Homma (1982) also reported abnormal egg shell formation by these females. All the defects are localized to the Mullerian duct system. Homozygous males and heterozygous females reproduce normally.

Pigmentation Restricted To Certain Feather Tracts

There are seven color variants in Japanese quail where the restriction of pigmentation affects only the primary pattern. These variants have partly white phenotypes.

Brown-splashed white (*p*). There have been several autosomal mutations in Japanese quail that result in mostly or partially white plumage. The first white plumage mutation found was the recessive brown-splashed white reported by Shimakura (1940), who gave it the gene symbol p because it affected pigmentation of the feathers. The homozygote is a white bird splashed with some darker feathers on the head, back, wings, and breast. The ventral surface of the bird is white. The darker feathers are either greyish brown without the wild-type markings (some wing coverts), or wild-type feathers with white edges and/or tips (back and body contours). The amount of dark feathers is variable among individuals. The iris of the mutants is much darker than wild-type. The beak is pale pink compared to the yellowish brown color of the wild-type. The down of chicks is white, faintly tinged on the back with grey.

Recessive white (*wh*). Subsequently, a similar mutation was reported by Roberts et al. (1978) who called it white (*w*), and by Somes (1979) who called it recessive white (*wh*). The latter symbol (*wh*) has been adopted for common use (Somes, 1984). This white mutation is carried in a commercial strain called English White.

The homozygote is also a white bird splashed with some wild-type feathers on the head and on the dorsal caudal, interscapular, and dorsopelvic tracts (Figure 13.1). The breast and ventral surface of the bird is totally white. The dark feathers do not differ from wild-type in color and markings. The amount of wild-type feathers on the head and back is quite variable among individuals. Chikamune (1984) reported that the amount of wild-type feathering in these white birds had very low heritability and maternal effects and therefore did not seem to be affected by modifying genes. The white birds have nonpigmented shanks and feet, but the iris and the pupil of the eye are pigmented as in wild-type. Unlike the *p/p* chicks, the down of *wh/wh* chicks is creamy yellow in color with a few black spots that will be replaced with wild-type feathers as adults.

Recessive white (*wh*) is epistatic to extended brown (*E*). Individuals with genotype *wh/wh E/-* are white splashed with brown feathers. A white bird homozygous for the wild-type allele at the *E* locus ($wh/wh\ e^+/e^+$) crossed to a wild-type individual ($Wh^+/Wh^+\ e^+/e^+$) will have progeny of wild-type plumage ($Wh^+/wh\ e^+/e^+$). However, if the individual is not homozygous for the wild-type allele at the *E* locus, the phenotypes will be very different. Individuals with genotype $Wh^+/wh\ E/E$ have the phenotype known as tuxedo. They are dark brown on the dorsal surface but white on the ventral surface. The area of white ranges from the total ventral surface to just a white band across the breast and a patch of white under the throat. Individuals of $Wh^+/wh\ E/e^+$ are known as shafted tuxedo. Their dorsal surface is shafted as in $Wh^+/Wh^+\ E/e^+$, but their ventral surface is white (Chikamune & Kanai, 1978; Truax & Johnson, 1979). Recessive white is also epistatic to yellow (*Y*). Birds with genotype $wh/wh\ Y/y^+$ are white splashed with yellow feathers (Chikamune & Kanai, 1978).

Panda (*s*). Another splashed-white mutation having a similar phenotype to *p* and *wh* was reported by Mizutani et al. (1974). It was said to be different from the *wh* mutation in that the feathers around the eyes of homozygotes are always dark, and thus the name panda. However, examination of panda quail maintained in Dr. Wakasugi's laboratory in Japan has revealed that the feathers around the eyes are not always dark (Cheng, unpublished) and the previous assumption may be a translation error. This recessive mutation is also different from *p* because the breast feathers and the primaries of panda are always white whereas the breast feathers of brown-splashed white are sometimes splashed with darker feathers and their primary wing feathers may be dark. In general, it was said that the panda has bigger areas of wild-type feathers on the back than either of the other splashed-white mutations. Panda chicks are cream-yellow with areas of wild-type down around the eye, on the head, back, tail, and wing secondaries and coverts. The adult phenotype of panda is very similar to that of *wh/wh*. Recent allelic tests with birds acquired from Dr. Wakasugi's laboratory showed that panda and recessive white are homologous (Cheng, unpublished). Ito et al.

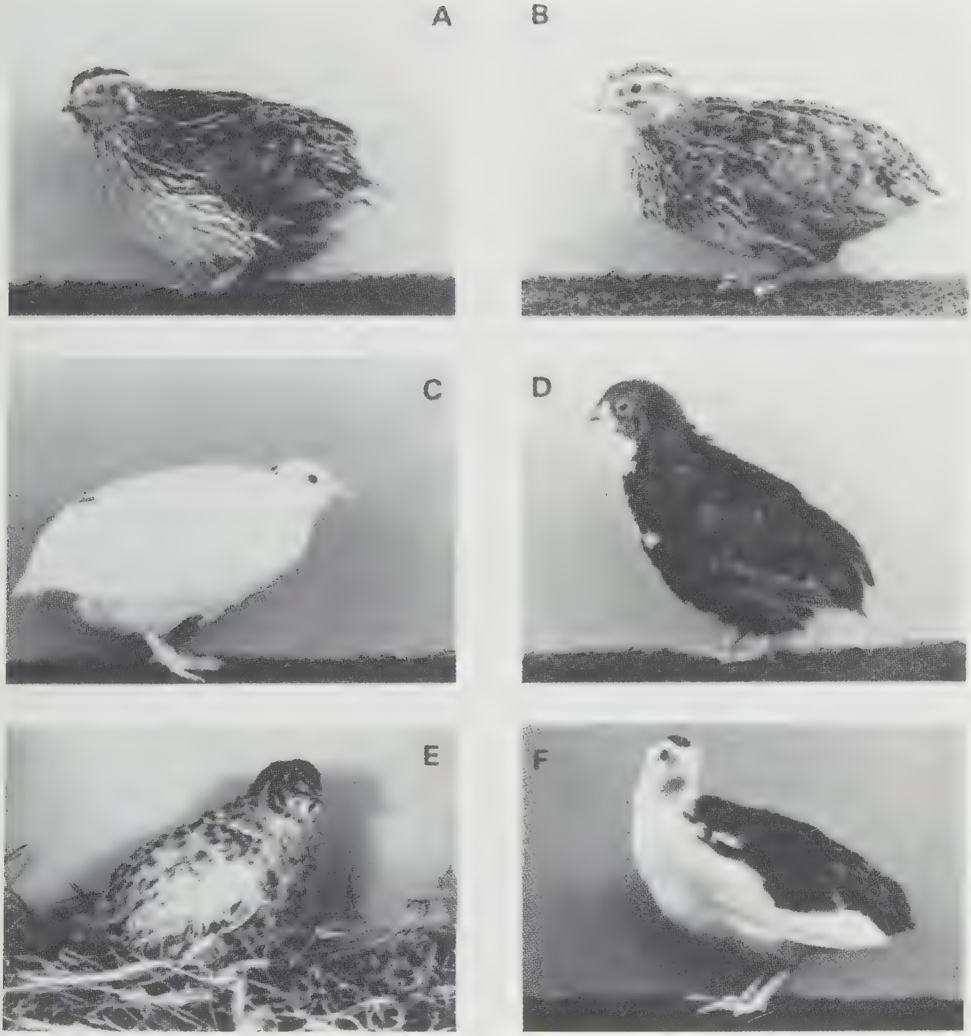

Figure 13.1. Plumage color mutants of Japanese quail: A - wild-type male, B - wild-type female, C - recessive white (*wh*), D - extended brown (*E*), E - red-head (e^{rh}), F - white-breasted (*wb*).

(1988a) conducted linkage tests and found independent segregation of *s* from esterase (*Es-D*), *Hb-1*, *Pgi*, and *Tf*. However, *s* is linked with serum albumin (*Alb*) at a recombination rate of 30 percent (Ito et al., 1988b).

Silver (*B*). Homma et al. (1969) found a white mutation which they called silver because although the homozygote (*B*/*B*) is pure white, the heterozygote (B/b^+) has greyish-white feathers and also shows the splashed or tuxedo pattern. This incompletely dominant mutation is associated with slow growth and sexual maturity when homozygous. Most homozygotes and some heterozygotes also have ring-retina in which the centre of the retina lacks pigment (Wakasugi and Kondo, 1973). Whether the retinal defect is a pleiotropic effect of the *B* locus or affected by another closely linked gene is uncertain. Silver segregates independently from *Es-D*, *Hb*, *Pgi*, and *Tf* (Ito et al., 1988a).

White-breasted (*wb*). Roberts et al. (1978) found a recessive mutation called white-breasted. Homozygotes are white-feathered on the face to just above the eyes, on the underside of the neck and the entire breast, and on the sternum up to and including the vent area. The primaries and most of the secondaries and their coverts are white (Figure 13.1). The remainder of the dorsal plumage is similar to wild-type color and pattern. The iris of the mutant is pigmented as in wild-type. Both sexes show the same plumage pattern. The chicks are cream-yellow with wild-type down on the dorsal surface.

White crescent (*cr*), white bib (*bi*). Roberts et al. (1978) also reported two traits involving areas of white feathers on a colored bird. The white crescent trait involves a white crescent-shaped band across the breast at the junction of the ventral cervical and the pectoral tracts. There is much variation in the size and shape of the white band. Segregation ratios indicated that the trait was likely caused by a recessive mutation (*cr*) but no further testing was carried out. The white beard trait, later renamed white bib (*bi*), involves white feathers found under the lower beak in the interramal tract. Again, the trait seemed to be affected by a single recessive mutation but there was no further testing.

White primaries (*wp*). Another trait that was reported to be affected by a single recessive gene was white primaries (*wp*) (Somes, 1984). While the trait appears to be recessive, it is not certain whether it is affected by only one gene (Cheng, unpublished). Selection for increased number of white primaries increased the frequency of birds with white feathers on the breast and ventral surface. Most birds with all white primaries become phenocopies of the white-breasted (*wb*/*wb*) mutant.

Amelanotics

Amelanotics are completely white individuals lacking pigmentation in all of the feather tracts.

White-feathered down (*c*). Several white mutations have been reported in the literature. The white-feathered down (*c*) found by Sittmann and Abplanalp (1965a) is recessive and *c*/*c* is lethal in the embryonic stage or within two days posthatch. The eyes of the mutants are normally pigmented.

Imperfect albinism (*al*). Imperfect albinism seems to be a commonly recurring mutation since it has been found in different populations (Wint, 1957; Lauber, 1964; Homma et al., 1968; Roberts, unpublished). Homozygous chicks have pink eyes and are light yellow to white in down color. Some chicks may show very faint longitudinal wild-type stripes on the back (Homma et al., 1968) while others may be totally void of wild-type markings (Lauber, 1964). The adult plumage is white with buff ghost-barring. Lauber (1964) examined feathers of imperfect albinos and found them completely lacking in melanin granules. She concluded that the ghost-barring is due to structural color only. Homma et al. (1968) reported that the ghost-barring was much darker in the summer and the whole bird appeared light brown, but the replacement feathers after the fall molt were white with very faint ghost-barring. Lauber and Vriend (1988, personal communication) found differences in retinal, pineal, and plasma melatonin levels between imperfect albino and wild-type birds under various light-dark regimens. The absence or presence of melanin granules in the feather barbules may depend on the lighting schedule that the birds are exposed to during feather growth. In addition, the intensity of the ghost-barring seems to respond to selection and may also be affected by modifying genes (Cheng, unpublished). Mérat et al. (1981) reported that albino quail have high early posthatch mortality (>30 percent) and depressed early growth. While age at first egg and egg number were not inferior to wild-type, feed consumption and egg weight were slightly less for albinos. Imperfect albino and dark-eyed dilute (al^D) mutants differ significantly between themselves and from the wild-type in susceptibility to glaucoma (Lauber and Cheng, 1989) and may be useful models for studying this disease.

Complete albino (*a*). Homma et al. (1968) reported a complete albinistic mutant and distinguished it from sex-linked imperfect albinism. Complete albinism is autosomal recessive. Homozygotes lack pigmentation through the whole body and do not show ghost-barring on feathers as do the imperfect albinos. Eyes of the complete albinos are ruby red and can be distinguished readily from those of imperfect albinos.

In reviewing the literature on plumage color mutations of Japanese quail, it has been a problem to decide whether some of the mutations may be actually the same but with different names. This problem is more serious in Japanese quail than in chickens because many reported mutations have been lost, or they are maintained in different countries. There is considerable difficulty in shipping birds across national borders to make the necessary testcrosses. In addition, there seemingly has been poor communication among quail geneticists in different countries.

Another phenomenon of theoretical interest is the relatively high frequency of spontaneously recurring mutations (eg. *al*, *Y*, *E*, *wh*, *cin*, etc.). If this is a reflection of the mutation rate occurring in the rest of the Japanese quail genome, then this high mutation rate may account for the severe inbreeding depression suffered by this species.

FEATHER GROWTH AND STRUCTURE

Quail feathers are very similar to chicken feathers. The natal down of wild-type quail chicks consists of a bud-like calamus and a cluster of barbs with plumulaceous barbules. The important features of a normal adult feather consist of the shaft, the barbs, and the barbules which have hooklets. The hooklets of the barbules interlock to form the vane of the feather (Lucas and Stettenheim, 1972).

There are six mutations found in Japanese quail involving abnormalities of growth or structure of the feather. Four of these mutations (rough-textured, short-barb, defective feathers, and porcupine) involve abnormalities in the regulation of development of feather parts. Not much information is available for the other two mutations, downless and ruffle. Two more mutations, ear tuft (*hfd*) (Tsudzuki and Wakasugi, 1988a) and throat tuft (Tsudzuki and Wakasugi, 1988b; Tsudzuki and Wakasugi, in press), will be discussed under variants of muscles and skeleton.

Rough-textured (*rt*). Roberts and Fulton (1979) reported that the rough-textured trait is caused by an autosomal recessive mutation *rt*. Homozygous (*rt*/*rt*) chicks have a matted wet-looking down that feels like fine sandpaper. When examined under a scanning electron microscope, the down feathers of mutant chicks were smaller than those of wild-type (Cheng and Brush, 1984). Some barbs were totally bundled by and fused with their own barbules with very few irregularly spaced barbules joining the barbs properly at an angle. The adult mutants also have the same rough-textured touch when handled. Their feathers are structurally modified and give the impression of a fine lace effect in the vanes. Unlike wild-type barbules which are tapering, the plates of the mutant barbules are wider at the distal end. The radial angles are larger than 90° so that the barbules point towards the shaft of the feather rather than to the tip of the barbs. Because the barbules point against the grain of the feather, they cause the rough-textured touch sensation. The mutant phenotype is thought to be caused by a misalignment of the feather parts during development causing the barbules to be joined to inappropriate barbs (Cheng and Brush, 1984). There is a 30 percent reduction in hatchability of eggs laid by *rt*/*rt* females. After comparing the feather proteins from rough-textured and three other feather structure mutants (porcupine, short-barb, and defective feathers) to those of the wild-type, Cheng and Brush (1984) concluded that in all four mutants, neither the structural genes nor the interaction of their products was abnormal. The structural abnormalities in these mutants might be caused instead by mutation of regulatory genes which produce flaws in the function or organization of the follicle.

Porcupine (*pc*). Porcupine is an autosomal recessive mutation discovered by Fulton et al. (1982b). The down of mutant chicks appears normal (Cheng and Brush, 1984). In adults, the shafts of mutant feathers appear normal, but after full emergence from the follicle the feather vane remains unfurled and is partly wrapped by the feather sheath in the proximal portion. This abnormal development results in feathers appearing like porcupine quills. The affected feathers are particularly noticeable on the wings and back. As the mutant birds grow older, the feather vane of the back feathers may partially unfurl. Mutations with a similar phenotype have been found in chickens (Waters, 1967) and pigeons (Cole and Hawkins, 1930). In quail, *pc*/*pc* females have lowered fertility and egg production. There is also a high percentage of thin-shelled and abnormally shaped eggs. Homozygous embryos and chicks up to one week old have only a 50 percent survival rate.

Short barb (*sb*). The most obvious effect of this autosomal recessive mutation is on the back feathers. The barbs on the leading edges of these feathers are extremely short, and have the appearance of having been broken (Figure 13.2). Other feathers of the body are also affected, but to a lesser degree (Fulton et al., 1982c). Microscopic examination reveals that before the barbs are fully developed, their tips are fused together to form a thickened ridge along the leading edge of the feather. In most feathers these thickened ridges are sheared from the vane due to normal wear and tear leaving a row of barbs with broken ends (Cheng and Brush, 1984). There is much variation among birds in the severity of the

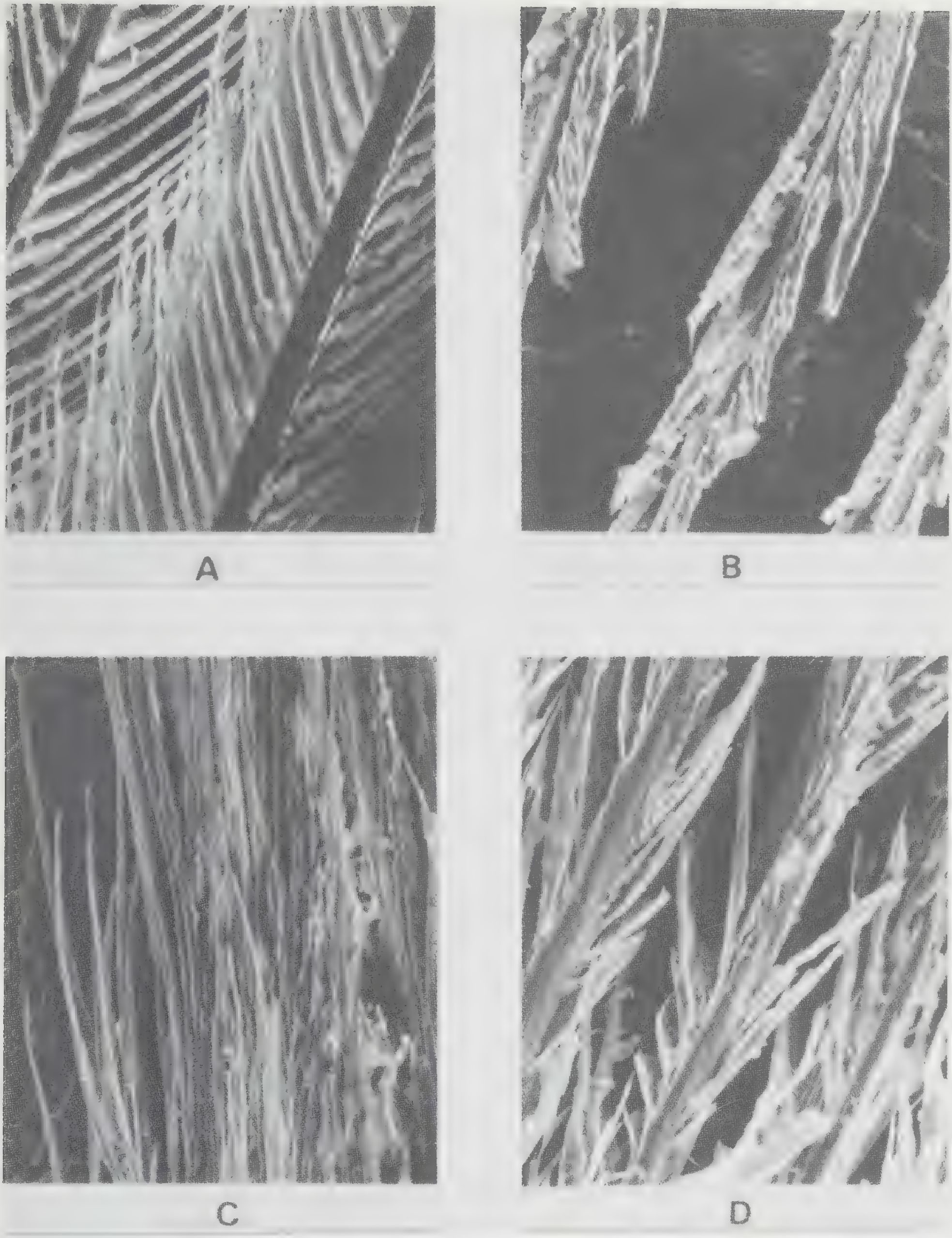

Figure 13.2. Feather structure mutants of Japanese quail. A - wild-type barbs and barbules (X100) of secondary remex. B - rought-rextured (*rt*) barbs and barbules (X100) of secondary remex showing curled barbules. C - short-barb (*sb*) back contour feather; magnified edge of vane (X150) showing fused barbules. D - defective feathers (*Df, mdf*); barbs and barbules (X100) of secondary remex showing broken barbules.

defect. Reproduction is not affected by this mutation.

Defective feathers (*Df* and *mdf*). The defective feather trait is controlled by an autosomal dominant gene (*Df*), whose manifestation is permitted by a recessive gene (*mdf*) at a second locus (Fulton et al., 1983). While *Df/df*$^+$ *mdf/mdf* individuals express the mutant trait, *Df/Df mdf/mdf* appears to be lethal. The down feathers of mutant chicks are short and sparse. Some severely affected chicks are totally bare. The feathers of adult mutants lack the interlocking web of the vane (Figure 13.2). In some individuals only the outer vanes of the feathers are affected. In others both the outer and the inner vanes are affected. Females seem to be more severely affected than males. Under magnification, the barbs are seen to be twisted in such a way that hooklets of one barb do not interlock with barbules of the adjacent barb. Many barbs are also broken across the base (Cheng and Brush, 1984). It is thought that in a mutant feather the cortical layer which provides it with a certain rigidity may be thinner than in a wild-type feather. This allows the barbs and barbules to be flexed out of alignment easily exposing those already fragile parts to additional wear and tear. Consequently there is more fraying and breaking of feathers in mutants.

Downless (*dl-1* and *dl-2*). The downless trait in quail was first observed by Savage and Collins (1971a). The trait seemed to be controlled by two autosomal recessive genes. Mutant chicks (*dl-1/dl-1 dl-2/dl-2*) had sparse scattering of down in all tracts except the capital, alar, dorsal lumbar, and pelvic which had a moderate coverage of down. Most downless chicks lived to three weeks of age but some reached maturity. Juvenile and adult plumages of the mutants were characterized by frayed and curled feathers. Most mutants lacked feathers on the ventral wing surface, sides of the body, thigh, and sternal tract. Downless males that survived to maturity had poor fertility, and females laid very few eggs. This mutant trait has not been maintained.

Ruffle (*rf*). The ruffle is an autosomal recessive mutation (Cheng, unpublished) found in the UBC-B (Alberta) wild-type line (Somes, 1988). In *rf/rf* individuals, the distal portion of the barbs of the inner vane is extremely soft and folds back onto the vane itself. There is variation in the severity of the defect and in the number of feathers affected. Occasionally wild-type individuals will also have one or two primaries showing this abnormality. Homozygotes (*rf/rf*) may have lower chick viability.

Feather formation is possibly the most complex developmental process occurring in the vertebrate skin (Sengel, 1976). Growth and morphogenesis involve a complex alignment of rows of epidermal cells to form the shaft, barbs, and barbules. Such processes have to be carefully regulated for the appropriate morphological units to form and interact. Feathers are composed almost exclusively of keratins, proteins which are unique in several respects and consist of a relatively large number of similar but not identical subunits (Busch and Brush, 1979). Details of the macromolecular organization and the mechanisms through which growth and alignment are regulated remain largely unknown. Many nutritional and stress factors can cause abnormalities in growth and structure of feathers (Murphy et al., 1988), some of which are similar to those caused by mutations. Studies utilizing feather structure mutants and involving these environmental factors should yield useful information regarding feather growth and development.

EGG SHELL COLOR VARIANTS

In various breeds of chickens, the brown egg-shell pigment which ranges in intensity from cream to almost purple is a hemoglobin porphyrin called protoporphyrin (also referred to as oöporphyrin). In some breeds that lay blue or green eggs, the blue pigment is composed of biliverdin and possibly a biliverdin-zinc complex. The outer surface of the Japanese quail egg-shell also consists of a base or ground color covering the entire surface. In addition, darker pigment patches are superimposed on the ground color resulting in a mottled appearance. Mottling is also found in turkey eggs although it is not as pronounced. Wild-type egg-shell ground color ranges from white to beige while the pigment patches are shades of brown or blue. In both the chicken and the Japanese quail, the egg-shell color is primarily the result of deposition of protoporphyrin and/or biliverdin pigments from specialized cells in the uterus onto the calcified shell in the last few hours prior to egg laying (Woodard and Mather, 1964; Poole, 1965). In Japanese quail, the superimposed dark pigment patches are deposited shortly before egg laying (Poole, 1965).

There are three reported shell color variations in Japanese quail: red, white, and blue.

White egg-shell (*we*). A white egg-shell mutant found by Poole (1964) is inherited as an autosomal recessive. The ground color of both the inner and outer surfaces of the mutant egg-shell is chalky white. Pigment patches on the outer surface of the shell, when present, appear as light red-brown to brown dots. While both protoporphyrin and biliverdin were found in the pigments of wild-type egg-shell, only protoporphyrin was found on egg-shells of *we* mutants (Poole, 1965). The lack of pigment deposits on the mutant egg-shell is due to the fact that the cytoplasm of apical cells lining the uterus of the mutant contain less protoporphyrin and biliverdin than corresponding cells of the wild-type (Poole, 1966, 1967), and probably not because the egg stayed in the uterus for a shorter time as is commonly believed. Mérat (1988, personal communication) found that the egg-shells of *we* mutants were 10 percent thinner than those from heterozygotes, but there was no significant increase in water loss during incubation. Females homozygous for *we* showed delayed age of first egg, weighed less at eight weeks of age, and had 15 percent lower hatchability.

Red egg-shell (*R*). A red egg-shell variant observed by Hardiman et al. (1975) is autosomal dominant. The mutant shell ground color varies from off-white to deep pink while the pigment patches range in color from light pink to dark red.

Blue egg-shell, celadon (*ce*). Hardiman et al. (1975) also observed a blue egg mutant in their stock of Japanese quail and made some preliminary testcrosses. Their data indicate a single locus mutation but the mode of inheritance was not clarified. The egg-shell ground color of these variants was off-white while the superimposed pigment patches ranged from medium to dark blue.

Ito et al. (1988c) found a Japanese quail hen that laid eggs with glossy pale blue shells lacking pigment patches. Genetic tests indicated that the trait was controlled by an autosomal recessive mutation. The mutation was named celadon (*ce*) because of the resemblance in color to that type of porcelain.

VARIANTS OF MUSCLES AND SKELETON

Abnormal skeletal and muscle conditions will be considered in this section. Many of these abnormal conditions originate from metabolic aberrations in early development of the embryo and these gross changes often lead to impaired viability or to lethality. Mutations involving muscular or skeletal abnormalities, but also resulting in lethality, will be discussed under Embryonic Lethals. Other conditions occur during rearing or adult periods. These conditions may be degenerative or disabling but are usually not lethal. Taylor (1974) commented that these variants frequently resulted from genotype x environment interactions and would be difficult to study by genetic procedures alone. Somes (1988) listed four such variants in Japanese quail: long beak, crooked neck, wry neck (Savage and Collins, 1971b), and crooked toes (Lucotte, 1974). The last two are polygenic traits and will not be discussed here. In addition, two newly discovered traits, ear tuft and throat tuft, will be discussed under this section because they involve defects in hyomandibular furrow closure in developing embryos.

Long beak (*lb*). Roberts (unpublished) observed birds with extra long beaks in a randombred population at University of British Columbia and tested for mode of inheritance of this trait. Initial testmatings seemed to indicate that the long beak trait was controlled by an autosomal recessive gene. Further testcrosses (Fulton and Cheng, unpublished) showed that while the trait was recessive, single gene inheritance could not be confirmed. Because the expression of long beak varied according to age and sex of the bird, and was easily confounded by shape, curvature, and breakage, quantifying of the trait was difficult.

Crooked neck. The crooked neck trait in quail (Dodson and Coleman, 1972) phenotypically resembles crooked neck in Brown Leghorn chickens (Jull and Quinn, 1931) but it is different from the crooked neck dwarf (Sittmann and Craig, 1965). Dodson and Coleman (1972) attempted but failed to determine the mode of inheritance for this trait. They have, however, limited the possibilities to an autosomal recessive mutation with low penetrance or to polygenic control. The mutant trait does not express itself until the chicks are six to seven days posthatch. There is a wide variation in the degree of severity among individuals but more males are affected than females. Although egg production is not affected, only a few crooked neck adults are able to mate and produce viable offspring.

Ear tuft (*hfd*). Tsudzuki and Wakasugi (1988a) reported a mutant with unilateral or bilateral ear tufts and irregularly shaped ear openings. Genetic tests indicated that the trait was autosomal recessive with about 35 percent penetrance. When five-day old homozygous mutant embryos were examined, however, 91 percent of the embryos showed incomplete and abnormal closure of the hyomandibular furrow. The frequency of abnormal five-day old embryos was slightly higher if both parents had ear tufts compared to matings of homozygous mutants with no ear tuft and normal ear openings. It is possible that there are modifying genes that are responsible for the repair of less severe hyomandibular defects during the later part of embryonic development (Tsudzuki and Wakasugi, 1988c). Because the mutation primarily affects the closure of the hyomandibular furrow, it was named *hfd* (hyomandibular furrow defects). The incomplete closure of the hyomandibular furrow may lead to the abnormal ear openings while peduncle growth in the depression inside the furrow probably results in ear tufts. The phenotype of ear tufts in quail is similar to the ear tufts trait in Araucana chickens, but the latter is controlled by a dominant mutation (*Et*) with homozygous lethality (Somes and Pabilonia, 1981; Pabilonia and Somes, 1981, 1983).

Throat tufts. Another trait affecting the hyomandibular furrow has been found in a strain of Japanese quail not related to that with the *hfd* mutation (Tsudzuki and Wakasugi, 1988b). A slightly different type of defective closure of the hyomandibular furrow and positions of peduncle growth resulted in unilateral or bilateral tufts of feather under the throat of the mutant. A higher percent of embryos with defects died shortly before hatching. Genetic tests indicated that the trait may be affected by an autosomal recessive gene with low penetrance, and the mutant gene may be allelic and dominant to the *hfd* gene. The evidence, however, is not conclusive and further testing is necessary (Tsudzuki and Wakasugi, in press).

EMBRYONIC LETHALS

Japanese quail are very susceptible to inbreeding depression (Sittmann et al., 1966b) and therefore it would be expected that lethal or semilethal mutations would be revealed when a population of quail is subjected to inbreeding (Lucotte, 1973). Kawahara (1976) compared the genetic load in wild and domestic Japanese quail and found that the lethal equivalent in domestic strains was lower than that of the wild strain examined. He concluded that some detrimental genes may have been eliminated in most domestic strains during their history of domestication, which is reasonable given the fact that lethal mutations are difficult to maintain and to study. Nevertheless, there are six lethal mutations that have been reported: abnormal head, arostroperocephaly, chondrodystrophy, chondrodystrophy-2, crooked neck dwarf, and micromelia. In addition, there are others that are lethal only when in homozygous state, and these have been discussed in other sections of this chapter.

Micromelia (*m*). This autosomal recessive lethal abnormality was observed by Hill et al. (1963) in three separate lines of Japanese quail maintained at the University of California-Davis. Affected embryos of both sexes died between 11 and 16 days of incubation. The head was short and broad with bulging eyes. The lower mandible was markedly reduced, and the upper beak was shortened and curved to give a parrot beak appearance. The wings and legs were set far apart. Long bones were reduced to half the normal length. These conditions resembled micromelia in chickens (Asmundson, 1942).

Crooked neck dwarf (*cn*). Sittmann and Craig (1965) found this autosomal recessive lethal in Japanese quail. It was very similar both in mode of inheritance and phenotype to the crooked neck dwarf found in chickens (Asmundson, 1945) and in turkeys (Asmundson and Pun, 1956). The mutant embryos had reduced body size, and necks that were rigid and bent sharply to one side, often to the right. The upper beak was usually shorter than the lower beak and the legs of the embryos were crossed at the tibiotarsal joint which was abnormally rigid. The age of death of mutant embryos ranged between 14 and 19 days of incubation and before pipping the air cell. It was suspected (Sittmann and Craig, 1965) that the rigidly bent neck and legs prevented the embryos from pipping. Unlike chicken and turkey crooked neck dwarfs, there was no abnormality of the digestive tract in the Japanese quail mutants.

Arostroperocephaly (*ar*). From a full-sib mating, Dodson and Coleman (1973) observed unhatched embryos with absence of all rostral membrane bone and nasal septum. These embryos also had eyeballs fused in the mid-line and no development of the maxillaries and palate. Subsequent mating tests showed that most embryos died around 14 days of incubation and that the trait was likely determined by an autosomal recessive gene.

Abnormal head (*ab*). Morse and Abplanalp (1970) reported the discovery of a head abnormality found in chicken x quail hybrids. The mutation affected the eyes, beak, and/or the anterior neural cord. The mutation was said to be recessive because the authors traced its origin to a pair mating of Japanese quail in 1962; presumably it had not expressed itself in the population because of its existence in a heterozygous state. In the chicken x quail hybrids, the trait became apparent either because there was no homologous locus on the chicken chromosome and therefore the quail gene became hemizygous in the hybrid genome, or because the quail gene was dominant over the homologous allele from the chicken genome. This mutation was classified by Somes (1988) as a lethal mutation but there was no mention of lethality in the original report.

Chondrodystrophy (*ch*). From one of the same lines of Japanese quail where Hill et al. (1963) found the micromelia (*m*) mutation, Collins et al. (1968) identified another embryonic lethal mutation that was quite similar. Although this new mutation also gave rise to parrot beak and short extremities, the mutants did not have the broad short head and bulging eyes nor the widely spaced wings and legs characteristic of the *m*/*m* mutants. Furthermore, with the new mutation, embryonic mortality occurred earlier around the eighth or ninth day of incubation. Mating tests indicated that the new mutation was autosomal recessive and it was called chondrodystrophy because of its similarity to the chondrodystrophy mutation in chickens (Lamoreux, 1942). Since the micromelia mutant had been lost, testcrossing for homology was not possible.

Chondrodystrophy-2 (*ch-2*). From a line of Japanese quail maintained at University of California-Davis, another chondrodystrophy mutation was found by Hermes et al. (1986). The new mutation was also characterized by having an overall shortening of the long bones, a reduced but parrot-like beak, and a flattened head. The shortened long bones of the wings and legs also showed a variable amount of bending. Testcrosses indicated that the new mutation was autosomal recessive and not homologous with the one found by Collins et al. (1968).

Deformed beak (*db*). An embryonic lethal mutation characterized by a very short upper beak frequently accompanied by encephalocele or protrusion of the brain through an open cranium has been found by Cheng (unpublished). Preliminary analyses indicate that the mutation is likely to be autosomal recessive. A large percentage of *db* embryos die early before phenotype can be identified, and others die at various stages throughout the incubation period.

NEUROLOGICAL MUTANTS

Individuals having neurological defects are usually disabled if not lethal, and are difficult to maintain and propagate for genetic testing. To date, only three single gene neurological mutants have been reported (*lo*, *sg*, and *dn*). A line of quail which carries a hereditary nervous disorder called myelin deficiency, has been maintained by the Nippon Institute for Biological Science in Japan since 1981 but it has not been described in the literature. Another neurological trait, back-drawer (Tsudzuki and Wakasugi, 1988d), seems to be controlled by two loci.

Congenital loco (*lo*). Knowlton (1929) first described an abnormality in chickens at hatching in which the head is directed backward, the beak pointed upward, and the chick's balancing mechanism is disturbed. He named it congenital loco. Since then the condition has been observed in several breeds of chickens, and poultrymen commonly use the term star-gazer to describe it. In Japanese quail, congenital loco (*lo*) and star-gazing (*sg*), are names given to two different mutations (Savage and Collins, 1972) although critical tests have not been performed to determine if the two mutations are homologous (see also star-gazing). Sittmann et al. (1965) first reported congenital loco in Japanese quail and found that although the homozygotes of this autosomal recessive mutation are semilethal, only a very small percentage reach sexual maturity. The trait is expressed in day-old quail chicks by lack of balance and in mature birds by pronounced excitability and rapid circling. Day-old chicks snap their head and neck backwards or to either side while rapidly shaking their head. Such movements often cause them to fall over. No signs of the condition are observed when the chicks are resting undisturbed. Macroscopic examination of the inner ear morphology of mutant chicks revealed extensive defects of the semicircular canals (Savage and Collins, 1972).

Star-gazing (*sg*). Sittmann and Abplanalp (1965b) made brief reference to this mutant trait in Japanese quail, but Savage and Collins (1972) analyzed the mode of inheritance and made comparisons between star-gazing and congenital loco. Star-gazer is also autosomal recessive, but the viability of star-gazer is much higher than that of congenital loco. Star-gazing is only observed infrequently at hatching and the behavioral abnormality is only apparent after the birds are three weeks old. Unlike congenital loco, there is no morphological abnormality of the inner ear of star-gazers. Many star-gazers, male and female, can mate and produce progeny.

Dark feather nervous disorder (*dn*). This autosomal recessive mutation found by Kawahara (1979) is characterized by cerebellar functional disorders such as fine tremor of the extremities and trunk, gait disturbance, and tumbling when the bird is active or excited (Ueda et al., 1979). The mutation is semilethal and survival up to 20 weeks of age is about 40 percent. Many birds die of dislocation of the hip joint. The homozygotes also have dark and frayed plumage that is different from the wild-type. Abnormalities in the organization and morphology of the Purkinje cells have been detected in the mutants. The mutation causes slower growth rate and sexual maturity, and lower egg production, fertility and hatchability.

Myelin deficiency (*myd*). This unreported autosomal recessive mutation was found by Dr. Makoto Mizutani of the Nippon Institute for Biological Science in Japan. Affected birds show trembling as newly-hatched chicks and the trembling continues through the bird's life (Mizutani, 1988, personal communication). Histological analyses reveal that the glial cells of the optic nerve are myelin deficient. The number and diameter of myelinated axons are also affected. More detailed information is not available. For homozygotes, fertility is 25 percent, hatchability of fertile eggs is about 40 percent, and posthatch mortality to six weeks of age is 80 percent.

Back-drawer (*bkd-1*, *bkd-2*). This behavioral mutant is characterized by crouching and bending the neck downward between the legs. The affected bird walks backwards (back-drawing) and is only able to move forward by tumbling and rolling (Tsudzuki and Wakasugi, 1988d). When the affected bird hops or jumps, it is also in a backwards direction. There is considerable variation in severity of expression. In its mild form, the affected bird is able to move in a normal fashion except for a slight but consistent downward bending of the neck. The trait may express itself at hatching. In this case it is always in its severest form and the chicks invariably die within a few days. Affected birds may also behave normally at hatching and start to show the abnormality between 20 to 60 days after hatching. Many affected males go through stages of mild to severe abnormality and then recover and behave normally. Others, mostly females, may remain in the severe stage and die. Preliminary histopathological examination of birds killed while they were at a stage of severe abnormality revealed abnormal organization of the Purkinje cells. Genetic tests indicated that the trait may be affected by two autosomal loci and that the trait expresses itself when both loci are homozygous recessive. The mutant trait is maintained by crossing affected males with phenotypically normal females from affected families.

METABOLIC AND DEVELOPMENTAL VARIANTS

Glycogenosis type-II (*glyII*). Generalized glycogenosis is an inherited glycogen storage disease reported in many species of animals (Nunoya et al., 1983). A mutant in Japanese quail that resembles glycogenosis type 2 in humans was found by Murakami et al. (1982). The condition appears between two and 12 weeks of age and tissue deposition of glycogen increases with age. By six months of age, affected quail show difficulty in raising their wings. Excessive accumulation of glycogen can be seen in the liver, heart, skeletal muscle and brain, apparently because of decreased acid maltase activity (Matsui et al., 1983; Nunoya et al., 1983). From enzyme analyses, it is suspected that the trait is affected by an autosomal recessive gene (Nunoya et al., 1983). The mutant may be useful as a model for the investigation of generalized glycogenosis in man.

Twinning (*Tw*). Extended pre-incubation storage of fertilized eggs has been known to cause abnormal development of embryos (Landauer, 1967). Partial and complete twinning from a single yolk is relatively rare in chickens and turkeys but seems to be more common in Japanese quail (Homma and Jinno, 1968). These abnormal developments were considered to be of environmental origin until Sittmann et al. (1971) examined twinning as an expression of either dam or sire genotype and conducted pedigree analyses on families that had instances of twinning. They concluded that the propensity to produce twin progeny in stored eggs of Japanese quail is inherited as an autosomal dominant trait. The expression of this trait may be modified by unknown environmental factors in addition to length of storage.

IMMUNOLOGICAL VARIANTS

There are variations among Japanese quail strains in susceptibility and resistance to specific pathogens (Morikawa et al., 1984). Selection for specific disease resistance can be effective (eg. the Brwinow strain in Poland, resistant to Newcastle disease after just three generations of selection. See also Takahashi et al., 1988).

Studies on blood typing in Japanese quail were limited in the early stages because of difficulties in obtaining specific antisera in adequate amounts (Mizutani et al., 1977a). The use of plant phytohemagglutinin (PHA) resulted in the detection of several blood types (Mizutani et al., 1977a). More blood groups were identified later using alloantiserum from Japanese quail and heteroantisera from rabbits and cows (Katoh and Wakasugi, 1980, 1981).

Sb agglutinogen inhibitor (*Sn*). PHA from extracts of soybean (*Glycine max*) agglutinates red blood cells of Japanese quail (Sb agglutinogen). However, red blood cells of laying females carrying the autosomal dominant *Sn* gene are not agglutinated by soybean PHA (Mizutani et al., 1977b). Males and nonlaying females carrying the gene are not affected and their red blood cells can still be agglutinated. When young quail or castrated males carrying the gene are injected with estrogen, the agglutinating ability of their red cells disappears. Estrogen treatment of sn^{+}/sn^{+} individuals does not change their agglutinating ability. When red cells from laying *Sn*/- females are treated with 0.5 percent trypsin solution, they are agglutinated by soybean PHA. Thus it seems that the inhibitor to soybean PHA agglutination of red blood cells, presumably the *Sn* gene product, is activated by estrogen but it is also trypsin sensitive.

Pn agglutinogen inhibitor (*pn*). PHA from peanut seeds (*Arachis hypogaea*) causes hemagglutination in Japanese quail (Pn agglutinogen). For *pn*/*pn* individuals, erythrocytes can be agglutinated in embryos from seven to nine days of incubation. After ten days of incubation, however, erythrocytes that will not agglutinate begin to appear, and by the time the chicks are one week posthatch, their erythrocytes are not agglutinated by peanut PHA. Mizutani et al. (1977c) proposed that the agglutination by peanut PHA in Japanese quail be treated as an autosomal dominant trait. Because erythrocytes from all Japanese quail can be agglutinated by peanut PHA during early embryonic stages, it would be more suitable to treat agglutinating ability as wild-type (Pn^{+}) and nonagglutinating as recessive (*pn*).

Red blood cell antigens (*Ea-A*, *Ea-B*, and *Ea-C*). Using antisera against Japanese quail red blood cells obtained from Japanese quail, rabbits, and Holstein cows, Katoh and Wakasugi (1980) detected three red blood cell antigen loci. Mating tests indicated that the three antigens were controlled by three separate loci. The frequencies of the three dominant alleles were also very different in the seven plumage color strains that were maintained in their laboratory. The authors initially named the three loci *QN*, *R*, and *H* reflecting the source of antisera used for detection. In a later paper (Katoh and Wakasugi, 1981) the authors changed the names of these loci to *A*, *B*, and *C*, respectively. According to Somes (1980) the proper gene symbols are *Ea-A*, *Ea-B*, and *Ea-C*, where *Ea* represents erythrocyte antigen. Strains of Japanese quail that are homozygous for each locus and recessive for the other two have been developed for immunogenetic studies (Katoh and Wakasugi, 1981).

Ly_1 and Ly_2 antigens (Ly^1, Ly^2, Ly^3). Using antisera obtained by immunizing Japanese quail with Japanese quail leukocytes, Katoh and Wakasugi (1981) detected two antigens that are present on red blood cells, peripheral leukocytes, and spleen cells. Genetic tests indicated that these two antigens are controlled by codominant alleles of a single autosomal locus. Simultaneously typing the various experimental strains with anti-A, anti-B and anti-C sera revealed that the presence of Ly_1 and A antigens are closely associated, an indication that the *Ly* locus may be part of the MHC in Japanese quail. There was also variation in the frequencies of the Ly^1 and Ly^2 alleles in different strains of quail. Recently a third allele, Ly^3, has been found (Mizutani, 1988, personal communication) but detailed information concerning it is not available.

POLYMORPHIC PROTEINS AND ENZYMES

Garcia (1983) reviewed information available on major proteins and enzymes from various tissues of Japanese quail and noted that of the 20 major proteins studied, 15 have been found to be polymorphic. Of 49 enzymes examined, 26 are polymorphic.

Proteins. Egg and blood proteins of Japanese quail have been examined by Baker and Manwell (1967) and Lucotte and Kaminski (1975a, 1975b). They have published composite electrophoretic diagrams of egg white and serum proteins which they found. Individual variations of nonenzymatic proteins are listed in Table 13.2.

Riboflavin-binding protein, ovalbumin, ovomacroglobulin, albumin (Lucotte and Kaminski, 1975b, 1977), ovalbumin-A_1 (Tanabe, 1980), and ovalbumin-A_3 (Baker and Manwell, 1967) show variation in staining intensity as detected on starch or acrylamide gel. In these cases three phenotypes are observable: heavily stained, lightly stained, and faintly stained/absent.

Other proteins listed in Table 13.2, except for ovomucoid and hemoglobin, are single locus traits under the control of autosomal loci with codominant alleles. Each allele codes for a protein band with different electrophoretic mobility and heterozygotes would therefore show two protein bands.

Bogard et al. (1980) showed that either serine, glycine, or an equimolar mixture of the two can exist at position 162 of ovomucoid isolated from Japanese quail. They accordingly proposed two ovomucoid structural alleles, Ovm^S and Ovm^G.

Hemoglobin of adult quail can be divided electrophoretically into a major and a minor component. Individual variation has not been observed for the major component but variations in the minor component have been observed in some populations. Three structural loci, α^D, α^A, and β, code for adult chicken hemoglobin (Keane et al., 1974). Adult hemoglobin of Japanese quail is also believed to be coded by three structural loci (Baker and Manwell, 1967): the major adult hemoglobin unique chain locus (monomorphic), the common adult hemoglobin chain locus (monomorphic), and the minor adult hemoglobin unique chain locus (polymorphic). Washburn et al. (1968) found two alleles at this polymorphic locus and named them m_1 and m_2. Maeda et al. (1975) also reported two alleles (*A* and *B*) for this locus which they named *Hb-1*. The gene symbols $Hb\text{-}1^A$ and $Hb\text{-}1^B$ have now been adopted (Somes, 1984). Maeda et al. (1975) found that $Hb\text{-}1^B/Hb\text{-}1^B$ individuals have slower early growth, higher early posthatch mortality, and lower egg production and fertility. Dimri et al. (1981) also reported that the $Hb\text{-}1^B$ allele is associated with lower hatchability. This may be the reason for the low frequency of the $Hb\text{-}1^B$ allele in most randombred populations (Maeda et al., 1975).

As in chickens and ring-necked pheasants, transferrin and conalbumin are controlled by the same locus (Kimura et al., 1977), whereas serum albumin and yolk albumin are controlled by another locus (Kimura et al., 1980b).

Enzymes. Individual variations of enzymatic proteins are listed in Table 13.3.

Lush (1966) reported an additive effect for alleles at the locus affecting catalase activity of quail albumin. Homozygotes for the allele Oct^L have a low catalase activity while homozygotes for the Oct^H allele have a high catalase activity. The catalase activity of heterozygotes is intermediate.

Other enzymes listed in Table 13.3, with the exception of Es-III, Es-5, Es-6, Es-7, Akp, and LDH, are single gene traits controlled by autosomal loci with codominant alleles.

Each of the four exceptional nonspecific esterases (Es-III, Es-5, Es-6, and Es-7) is controlled by a single locus with a dominant and a recessive allele designated *A* and *o*, respectively. Homozygotes for the *o* allele do not show any esterase band on the gel.

The alkaline phosphatase allele Akp^{90} is dominant to the allele Akp^{89} (Savage and Collins, 1971c). Mobility of the phosphatase band 89 is slower towards the anode than that of band 90. Heterozygotes have only one phosphatase band 90.

Manwell and Baker (1969) found that red cell lactate dehydrogenase (LDH) was controlled by two loci, *Ldh-H* and *Ldh-M*, and reported variations in staining intensity of the five enzymatic bands. Kimura et al. (1984) also found that one of the LDH loci was

Table 13.2. Polymorphic proteins of Japanese quail.

Protein	Origin	Gene symbol[1]	Reference
Albumin	serum	$Alb^{Q1, Q2}$	Haley (1965)
		F, m, f	Lucotte and Kaminski (1975b)
Albumin	egg yolk	$Alb^{A, B}$	Kimura et al. (1980b)
Globulin	serum	$Gl^{F, S}$	Lucotte and Kaminski (1975b)
Hemoglobin	red cells	$Hb\text{-}1^{A, B}$	Maeda et al. (1975)
		(m_1, m_2)	Washburn et al. (1968)
		(2 alleles)	Baker and Manwell (1967)
Lysozyme	egg white	$G_1^{F, S}$	Baker and Manwell (1967)
Major livetin protein	egg yolk	(2 alleles)	Baker and Manwell (1967)
Minor livetin protein	egg yolk	(2 alleles)	Baker and Manwell (1967)
Ovalbumin	egg white	$Ov^{A, B}$	Csuka and Novy (1974); Garcia et al. (1977)
		F, m, f	Lucotte and Kaminski (1975b)
Ovalbumin-A_1	egg white	$Ov\text{-}1^{A, B}$	Tanabe (1980)
Ovalbumin-A_3	egg white	$Ov\text{-}3^{A, B}$	Baker and Manwell (1967)
Ovoglobulin G_2	egg white	$Ovg2^{F, S}$	Lucotte and Kaminski (1975b)
Ovoglobulin G_3	egg white	$Ovg3^{F, S}$	Lucotte and Kaminski (1975b)
Ovomacroglobulin	egg white	$Omg^{F,M,S}$	Baker and Manwell (1967)
		F, m, f	Lucotte and Kaminski (1977)
Ovomucoid	egg white	$Ovm^{S, G}$	Bogard et al. (1980)
Prealbumin-1	egg white	$Pa\text{-}1^{A\text{-}C}$	Tanabe (1980)
Prealbumin-2	egg yolk and serum	$Pa\text{-}2^{A, B}$	Tanabe and Ogawa (1982)
	serum	(F, S)	Lucotte and Kaminski (1975b)
Prealbumin-3	egg yolk and serum	$Pa\text{-}3^{A\text{-}D}$	Tanabe et al. (1981)
Prealbumin-4	egg yolk	$Pa\text{-}4^{A, B}$	Tanabe et al. (1981)
Pretransferrin	egg yolk	$Ptf^{A, B}$	Tanabe et al. (1981)
	serum	(F, S)	Lucotte and Kaminski (1975b)
Postalbumin	egg yolk	$Poa\text{-}1^{A,B}$	Tanabe et al. (1981)
	egg white	(F, S)	Baker and Manwell (1967)
Riboflavin-binding protein	egg white	F, m, f	Lucotte and Kaminski (1975b)
Transferrin	egg white and serum	$Tf^{A\text{-}C}$	Kimura et al. (1977)
X-Protein	egg white	(F, M, S)	Baker and Manwell (1967)
Y-protein	egg white	(F, M, S)	Baker and Manwell (1967)

[1] Symbols not in italics represent phenotypes; symbols in parentheses represent original gene symbols.

Table 13.3. Polymorphic enzymes of Japanese quail.

Enzyme (E.C. No.)[1]	Origin	Gene symbol[2]	Reference
Acid phosphatase-1 (3.1.3.2)	liver	*Acp-1*^{A,B}	Somes (1988)
Acid phosphatase-2	liver	*Acp-2*^{A,B}	Maeda et al. (1976)
Alcohol dehydrogenase (1.1.1.1)	liver	*Adh*$^{A\text{-}C}$	Castro-Sierra and Ohno (1968)
	serum	*(1,2)*	Lucotte and Kaminski (1975a)
Alkaline phosphatase (3.1.3.1)	serum	*Akp*^{90,89}	Savage et al. (1971c)
		(1, 2)	Lucotte and Kaminski (1975a)
Alkaline phosphatase-2	serum	*Akp-2*$^{A\text{-}C}$	Maeda et al. (1972)
Alkaline phosphatase-5	serum	*Akp-5*^{F,S}	Somes (1988)
Amylase (3.2.1.1)	serum	*Amy-1*^{A,B}	Watanabe et al. (1975)
	pancreas	*Amy-2*^{a,b}	Watanabe and Wakasugi (1978)
	egg white	*(A, B)*	Csuka and Novy (1974)
Aspartate aminotransferase-I (2.6.1.1)	liver	*Aat-I*^{A,B}	Kimura et al. (1982)
Catalase (1.11.1.6)	egg white	*Oct*^{H,L}	Lush (1966)
	liver	*Ct*^{F,S}	Kimura et al. (1978a)
Esterase (3.1._._)	liver	*(Es*^{F,S}*)*	Kimura et al. (1971)
		(Es^{C}*)*	Kimura et al. (1984)
	red cells	*Es-1*^{F,S}	Baker and Manwell (1967)
	liver	*Es-2*$^{A\text{-}C}$	Hashiguchi et al. (1978)
	brain	*(Es-II*^{A,B}*)*	Kimura et al. (1978b)
	red cells	*(Es-II*^{F,S}*)*	Baker and Manwell (1967)
	red cells	*Es-3*^{F,S}	Baker and Manwell (1967)
	pancreas	*Es-4*^{A,B}	Hashiguchi et al. (1976)
		Es-4^{C}	Kimura et al. (1982)
	serum	*(Es-IV*^{F,S}*)*	Lucotte and Kaminski (1975a)
	serum	*Es-5*^{A,o}	Hashiguchi et al. (1981)
		(Es-V^{F,S}*)*	Lucotte and Kaminski (1975a)
	serum	*Es-6*^{A,o}	Hashiguchi et al. (1981)
	serum	*Es-7*^{A,o}	Hashiguchi et al. (1981)
	red cells	*Es-8*^{A,B}	Hashiguchi et al. (1980)
Esterase	serum	*(Es-II*^{1,2}*)*	Lucotte and Kaminski (1975a)
	brain	*Es-III*^{A,o}	Kimura et al. (1978b)
	egg yolk	*(2 alleles)*	Baker and Manwell (1967)
	serum	*Es-D*^{F,S}	Watanabe et al. (1977)
Glutamate dehydrogenase (1.4.1.2)	liver	*Gdh*^{A,B}	Kimura et al. (1980a)
Glycerolphosphate dehydrogenase (1.1.1.8)	liver	*α-Gpdh*^{A,B}	Kimura et al. (1984)
Isocitrate dehydrogenase-I (1.1.1.42)	liver	*Icdh-I*^{A,B}	Kimura et al. (1984)
Isocitrate dehydrogenase-II	liver	*Icdh-II*^{A,B}	Kimura et al. (1984)
Lactate dehydrogenase (1.1.1.27)	red cells	*Ldh*^{F,S}	Manwell and Baker (1969)
Lactate dehydrogenase-H	heart	*Ldh-H*$^{A\text{-}C}$	Kimura et al. (1984)

Continued ...

Table 13.3. Polymorphic enzymes of Japanese quail. (Continued)

Enzyme (E.C. No.)[1]	Origin	Gene symbol[2]	Reference
Malate dehydrogenase-I (1.1.1.40)	liver	$Me\text{-}I^{A,B}$	Kimura et al. (1980a)
Mannose phosphate isomerase-I (5.3.1.8)	liver	$Mpi\text{-}I^{A\text{-}D}$	Kimura et al. (1980a, 1984)
Glucose-6-phosphate dehydrogenase (1.1.1.49)	red cells	$Pdh^{F,S}$	Manwell and Baker (1969)
Phosphogluconate dehydrogenase (1.1.1.44)	red cells	$Pgd^{A\text{-}D}$	Ohno et al. (1968)
Phosphoglucose isomerase (5.3.1.9)	red cells	$Pgi^{F,S}$	Juneja and Wilhelmsson (1975)
		Pgi^{S2}	Kimura et al. (1980c)
Phosphoglucomutase (2.7.5.1)	liver	$Pgm^{A,B}$	Kimura et al. (1982)
Sorbitol dehydrogenase (1.1.1.14)	liver	$Sdh^{A,B}$	Kimura et al. (1980b)

[1] Enzyme Commission classification number.

[2] Symbols in parentheses represent original gene symbols proposed.

polymorphic ($Ldh\text{-}H^A$, $Ldh\text{-}H^B$, and $Ldh\text{-}H^C$). They reported that most birds displayed a one-banded electrophoretic pattern for cardiac muscle LDH but some individuals had five-banded patterns specific to tetrameric proteins.

SUMMARY

The development of qualitative genetics can be divided into two phases: an initial phase of description and inventory, and a later phase of association and integration. During the initial phase, mutations are detected, the mode of inheritance is determined, and they are classified and catalogued. Those that can be maintained can be studied more closely during a second phase when linkage relationships, allelism, pleiotropic and epistatic effects, gene regulation, biochemical pathways, and physiology of gene products can be examined in association with the rest of the genome. In chickens, many mutations that were maintained and studied became useful research models for information about development, regulation, and functions of various body systems. This information is not only useful for improving poultry production but also useful in medicine and other areas of applied biology.

Because the Japanese quail is not as common as chickens in terms of poultry production, and because of the assumption that what is true for the chicken is true for the quail, research efforts in qualitative genetics of Japanese quail have been lacking, and the paucity of information about the quail genome has become obvious. Most of the available information can be summarized in a single chapter. This trend, however, is changing with the increasing popularity of Japanese quail as a research animal, and the development of various specialized strains for research purposes. With recent advances in molecular biology, many useful tools are now available for studying qualitative traits (eg. McNeish et al., 1988). An increasing rate of information accumulation can be foreseen in the future.

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Chapter 14. MUTATIONS AND MAJOR VARIANTS IN GUINEA FOWL

Ralph G. Somes, Jr.

The guinea fowl has been the basis of several qualitative genetic studies. The various plumage color variants and several feather mutants are the only visible traits that have been studied and recorded. Several biochemical polymorphisms have also been briefly looked at. The inheritance of these single gene traits is summarized in Table 14.1.

FEATHER COLOR TRAITS

Until recently, most of the genetic studies dealing with mutants of guinea fowl plumage color were those of the Italian geneticist Dr. Alexandro Ghigi. He presented a summary of the inheritance of guinea fowl plumage colors at the 2nd World's Poultry Congress in Barcelona (Ghigi, 1924). What follows is a summary of that paper, a later paper by Ghigi (1966), and this author's recent six years of experience with these plumage colors (Somes, 1988).

Four different plumage color loci are involved in the production of 17 different phenotypes (Table 14.2). The genes involved are covered on the following pages. The names of the phenotypes followed by (Ghigi) are the names that he used for these color types. The other names are those in more common use today.

Table 14.1. Genetic traits of the guinea fowl.

Trait	Gene symbol	Mode of inheritance
	Visible traits	
Absence of pearl markings	m, M^+ *	recessive
Blue dilution	i, I^+	autosomal recessive
Buff-dun	d, D^+	autosomal recessive
White	W, w^+	incompletely dominant
Aprotopteria	a, A^+ *	autosomal recessive
Sex-linked feathering	k	sex-linked
	Biochemical polymorphisms	
Mannosephosphate isomerase	$Mpi\text{-}1^A$, $Mpi\text{-}1^B$	autosomal codominant
Serum albumin	Alb^A, Alb^B	autosomal codominant
Serum amylase	Amy^A, Amy^B, Amy^C	autosomal codominant
Blood group	Gf, gf^+	autosomal dominant
Transferrin	Tf^A, Tf^B, Tf^C	codominant
Cytoplasmic aconitase	$Acon^B$	sex-linked
Ovalbumin	Ov^A, Ov^B	codominant
G_2 ovoglobulin	G_2^A, G_2^B	codominant
G_3 ovoglobulin	G_3^A, G_3^B	codominant

* Proposed gene symbols.

Grey (Ghigi), pearl, pearl grey, speckled or wild-type. The wild-type plumage color is that of the wild helmeted guinea fowl. This plumage color consists of white markings or pearls evenly spaced on an otherwise dark grey or violet-black background. The keets are reddish-brown with five black streaks on the head, the middle one being very large. It is against this color variety that the inheritance of the mutant loci was studied.

Violet (Ghigi) or royal purple (m). This phenotype consists of the dark intense violet-black ground color of the wild-type but with most of the white pearl markings absent, only a few being left on the body, sides and under the wings (Figure 14.1). Keets of this variety are white in the lower parts and the wings, while the dorsal surface is pale fawn and the middle head streak is much smaller and wavy compared to that of pearl keets (Figure 14.2). This phenotype results from the action of a recessive gene which prevents the production of what Ghigi called the 'margarogene factor (pearl producer)'. Thus pearled birds of any ground color are of either the genotypes M^+/M^+ *or* M^+/m and those lacking the pearl spots are m/m.

Lilac (Ghigi), lavender or light grey (i). Birds of this phenotype are a clear slaty blue color uniformly flecked with white pearl markings similar to those of the grey variety. Keets are a bright ash color with darker head streaks of the form and type of the grey keet. This dilution gene, i, is autosomal and recessive to its dominant allele I^+ for color intensity.

Ghigi (1924) reported on a sky blue color at the 2nd World's Poultry Congress. He obtained this new color as the doubly homozygous recessive from the F_2 generation of a lilac x violet cross. Birds of this color type are several shades of blue with the white pearls absent from most of the body. Keets are a bright ash color, with wings and underparts

Table 14.2. Guinea fowl plumage color phenotypes and genotypes.

Phenotype	Genotype
Grey, pearl grey, pearl, speckled, wild-type	$M^+/M^+ I^+I^+ D^+/D^+ w^+/w^+$
Violet, royal purple	$m/m I^+/I^+ D^+/D^+ w^+/w^+$
Lilac, lavender, light grey	$M^+/M^+ i/i D^+/D^+ w^+/w^+$
Sky blue, coral blue, blue coral	$m/m i/i D^+/D^+ w^+/w^+$
Chamois, dundotte, buff dundotte	$M^+/M^+ I^+/I^+ d/d w^+/w^+$
Buff	$m/m I^+/I^+ d/d w^+/w^+$
Porcelain	$m/m I^+/I^+ d/d w^+/w^+$
Opaline	$m/m i/i d/d w^+/w^+$
White	$-/- -/- -/- W/W$
Splashed, white-breasted pearl	$M^+/M^+ I^+/I^+ D^+/D^+ W/w^+$
Lakenpur, white-breasted purple	$m/m I^+/I^+ D^+/D^+ W/w^+$
Silverwing	$M^+/M^+ i/i D^+/D^+ W/w^+$
Coral white	$m/m i/i D^+/D^+ W/w^+$
Dundotte white	$M^+/M^+ I^+/I^+ d/d W/w^+$
Buff white	$m/m I^+/I^+ d/d W/w^+$
Porcelain	$M^+/M^+ i/i d/d W/w^+$
Opal white	$m/m i/i d/d W/w^+$

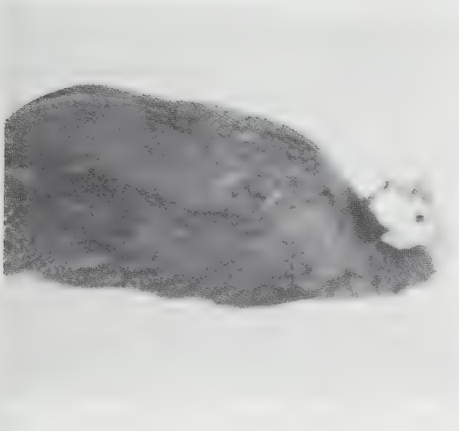

Figure 14.1. Adult guinea fowl homozygous for *m* gene which removes most of pearl markings.

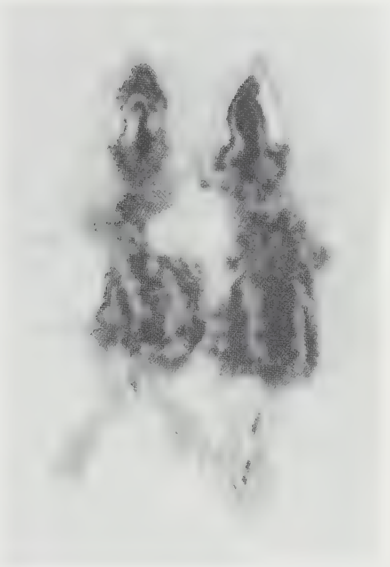

Figure 14.2. Day-old guinea fowl chicks with distinct head streaks characteristic of the presence of pearling (right), and the smaller wavy head streaks when pearling is absent (left).

white. Head streaks are darker but narrow and wavy. This color type is generally known as coral blue or blue coral at the present time.

Chamois (Ghigi), dundotte or buff dundotte (*d*). This variety has a buff ground color with white pearl markings covering the surface. The shade of buff can vary from extremely light, almost white, to as dark as the Rhode Island Red chicken. Keets are light buff or yellowish-white with darker head streaks in the form characteristic of the presence of pearl markings. Ghigi (1924) reported that he had not worked out the inheritance of this trait but knew that it was homozygous and different from the white guinea fowl. Somes (1988) has reported that the basic buff coloration was due to a single autosomal recessive gene to which he assigned the symbol *d*. He did not try to determine the cause of the wide variation in intensity that was observed among individuals of this phenotype.

The nonpearl-spotted counterpart of the dundotte is called buff. Birds are a more or less even shade of buff, with the pearl markings being absent. Buff coloration varies from light to dark as in the case of the dundotte.

Somes (1988), in his study of the buff-dun gene, crossed birds homozygous for the blue dilution gene with birds homozygous for the buff-dun gene. In the F_2 generation a new phenotype was produced which he referred to as blue-buff. This phenotype was the result of double homozygosity at the *i/i* and *d/d* loci. Birds of this coloration looked like buff birds with a very light blue wash over all their body except the neck. The neck remained buff. White pearls were present or absent depending upon the separate segregation of the *m* gene. Deeper colored birds were attractive, but some were so light that they could only be classified under very good light. These phenotypes are apparently the ones that

Greenwood (1987) refers to as opaline and porcelain.

White (W). The white variety of guinea fowl completely lacks pigmentation. The keets are also completely white. This phenotype is due to homozygosity of an incompletely dominant gene, W. Heterozygosity of this gene (W/w^+) produces a variegated pattern in which the dorsal surfaces are always colored, while the ventral surfaces are a more or less diffused white, as are also the tips of the wings. The white may be limited to a central spot on the breast and the flight feathers, or it can extend to cover all the breast and abdomen as well as the sides and a good part of the secondary wing feathers. White is never seen on the dorsal surfaces in heterozygotes (Figure 14.3).

The heterozygous white genotype (W/w^+) can express itself on any of the previously mentioned phenotypes. The dorsal surfaces are the mutant coloration, while the ventral surfaces are white. All of these phenotypes have been produced and they along with their respective genotypes are summarized in Table 14.2. The names assigned to these phenotypes were obtained from several sources (Ghigi, 1924; Greenwood, 1987; Van Hoesen and Stromberg, 1975). It is possible that some of these may also be known by other names.

Other colors. Greenwood (1987) referred to two other phenotypes which may represent some as yet uninvestigated plumage color loci. He mentioned a pied phenotype which could be combined with any of the other color phenotypes. This pied was unlike the somewhat fixed markings of the heterozygous white (W/w^+). Pied birds had splashes of white throughout their plumage, with no two birds looking alike. Greenwood (1987) claimed that the condition was recessive and did breed true. The second trait was one that he had not seen, but which had been described to him. It was black and had a much more even black coloring to its plumage than the usual royal purples.

FEATHER STRUCTURE AND GROWTH TRAITS

Aprotopteria (a). Only one inherited trait dealing with feather structure has been reported for the guinea fowl. McCrady (1932) described a so-called 'penguin' guinea fowl in which the flight feathers, the remiges, and the tail feathers, the rectrices, were absent. All other feathers were normal. The lack of flight feathers made the birds appear as if they were wingless; however, the wings were present and their skeletal structure was normal. Examination of the axial skeletion showed a normal number of synsacro-caudal and free caudal vertebrae, but the pygostyle was reduced in size by about 50 percent. Because of the extremely small number of birds available for study, the real significance and constancy of these skeletal observations remained unknown (Figure 14.4).

Three generations of breeding data demonstrated that this feather condition resulted from an autosomal recessive gene. Because the expression of this gene was limited to only the flight and tail feathers, McCrady (1932) proposed the name aprotopteria. He did not assign a gene symbol to the condition, but did use the letters A and a in describing its inheritance. In this case, these would be appropriate symbols and thus it is proposed here that they be used for this trait. Thus a/a is the aprotopterous condition and A^+/A^+ and A^+/a is the normal feathered wild-type.

Sex-linked feathering (k). The equivalent of the domestic fowl k gene, which affects feather growth rate, has been studied in the guinea fowl (Micek and Malik, 1970). In comparing remex length between males and females at 4, 8 and 12 days of age, significant differences were found. At 4 days of age, only 25 percent of the males but 68 percent of the females showed remex development. At 8 days, remiges of male keets averaged 25.05 mm vs. 32.00 mm for females, while at 12 days, they were 38.52 mm for males and 41.29 mm for females. Thus it appeared that a locus similar to the *k* locus of the chicken is present in the guinea fowl.

BIOCHEMICAL POLYMORPHISMS

A number of studies have been undertaken in the guinea fowl to examine blood, tissue and egg proteins for polymorphisms, and several protein variants have been shown to be under genetic control.

Mannosephosphate isomerase ($Mpi\text{-}1^A$, $Mpi\text{-}1^B$). Shiraishi and Hirai (1983) were able using electrophoresis to show the presence of genetic variants in guinea fowl liver and pancreas mannosephosphate isomerase. Each of their birds was of one of three types (A, B, or AB). Results from their mating experiments indicated that two codominant alleles were present at a single autosomal locus. The gene symbols $Mpi\text{-}1^A$ and $Mpi\text{-}1^B$ were proposed.

Serum albumin (Alb^A, Alb^B). The serum of one wild and two domestic strains of guinea fowl were examined by means of starch-gel and polyacrylamide-gel electrophoresis for variant serum isozyme patterns (Shiraishi et al., 1978). Three phenotypes (A, B, AB) were evident, with no birds showing other than one. Breeding tests indicated the presence of two codominant genes at a single autosomal locus, Alb^A and Alb^B.

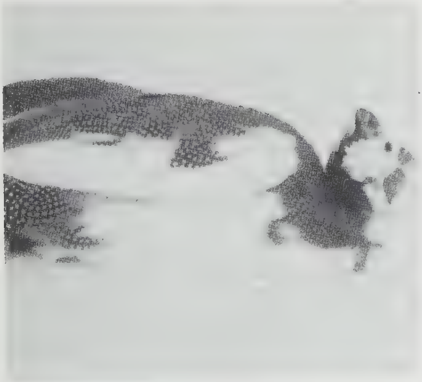

Figure 14.3. Adult guinea fowl heterozygous for the white gene, W/w^+. Dorsal surface has the wild-type pearl markings.

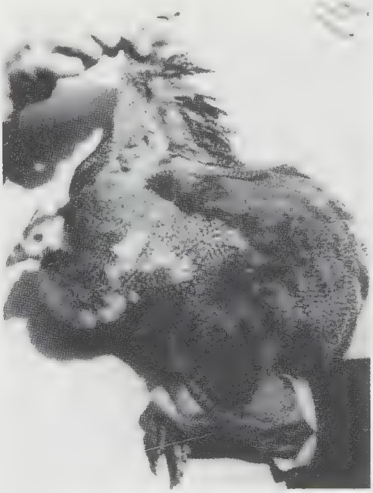

Figure 14.4. An aprotopterous cock nine months of age. The remiges and rectrices are missing. (From McCrady in J. Hered. 23:201, 1932).

Serum amylase (Amy^A, Amy^B, Amy^C). Five phenotypes of serum amylase (A, B, C, AB, BC) were electrophoretically distinguished from the same group of guinea fowl as were used in the serum albumin study (Shiraishi et al., 1978). The AC phenotype was not detected. Breeding tests among these birds showed that the variant patterns resulted from three codominant genes at a single autosomal locus, Amy^A, Amy^B and Amy^C.

Blood groups (*Gf*, *gf*). An erythrocyte agglutinin which was type-specific to guinea fowl erythrocytes was examined in four guinea fowl strains (Shiraishi et al., 1979a). Breeding tests showed that an autosomal dominant gene at a single locus, *Gf*, was responsible for the presence of the agglutinin while its recessive allele *gf* controlled its absence. The much lower gene frequency of the *Gf* allele in all but one strain would seem to indicate that *gf* is the wild-type.

The same agglutinin was also detected in 50 guinea fowl x chicken hybrids (Shiraishi et al., 1979b). Seventeen of the hybrids carried the *Gf* allele and 33 the gf^+ allele. The *Gf* type was further serologically classified into Gf_I and $Gf_I I$ subtypes. Only guinea fowl had the Gf_I subtype, but both guinea fowl and their hybrids had the $Gf_I I$ type.

Transferrin (Tf^A, Tf^B, Tf^C). Guinea fowl serum transferrin was compared via starch-gel electrophoresis with that of chicken and guinea fowl x chicken hybrids (Croizier, 1967). Guinea fowl transferrin patterns were quite different from those of the chicken. The guinea fowl had three bands, none of which were comparable to either the Tf^A or Tf^B patterns of the chicken. In the hybrid, the slowest migrating band of the guinea fowl was absent from the hybrid serum pattern.

Kierek-Jaszczuk (1976) examined 233 guinea fowl for their transferrin type and found four patterns, BC, AB, BB and AC. The AA and CC types were not present in their sample. They proposed that this variation resulted from three codominant genes at a single locus, Tf^A, Tf^B and Tf^C.

Cytoplasmic aconitase ($Acon^B$). Although no polymorphisms have been shown in the guinea fowl for cytoplasmic aconitase, it has been shown that its pattern was different than that of the chicken and that the locus controlling this enzyme was located on the sex chromosome (Baverstock et al., 1982). Electrophoretically the guinea fowl aconitase isozyme migrated much more anodally than that of the chicken. The type characteristic of the chicken was designated as type A, $Acon^A$, and that of the guinea fowl as type B, $Acon^B$. When the cytoplasmic fractions from hybrids produced between female guinea fowl and male chickens were examined for aconitase type, the results suggested that the *Acon* locus was on the sex chromosome. Male hybrids showed only the $Acon^A/Acon^B$ type while females showed only the $Acon^A$ type. These results were similar to those that would be expected if segregation involved a locus on the sex chromosome.

Egg albumin proteins (Ov^A, Ov^B; $G_2{}^A, G_2{}^B$; $G_3{}^A, G_3{}^B$). Valenta and Mikolasek (1971) examined three albumin proteins from each of 140 guinea fowl for polymorphisms. For each of the three proteins, ovalbumin, G_2 ovoglobulin and G_3 ovoglobulin, they were able to detect three phenotypes, A, B, and AB. Thus it would appear that the variations in segregation of each of these proteins results from the presence of two codominant genes at each of these three loci. Ovalbumin is controlled by Ov^A and Ov^B, G_2 ovoglobulin by $G_2{}^A$ and $G_2{}^B$, and G_3 ovoglobulin by $G_3{}^A$ and $G_3{}^B$.

Other possible polymorphisms. Sato et al. (1967) in a comparative study of serum proteins of guinea fowl, domestic fowl and their hybrids showed, using immunoelectrophoresis, that each breed had a single precipitin line but that the chicken line migrated more rapidly. The hybrids had two lines, thus the guinea fowl differed from the chicken. Two codominant alleles were suggested to explain these results. They also stated that the α 1-lipoprotein type was different between the guinea fowl and the chicken, and that the hybrid between these species was similar to the chicken type. Thus the chicken gene for this trait appeared to be dominant to that of the guinea fowl. These same authors also stated that immunoglobulin G was different between the species. Hilgert and Vojtiskova (1959) were able to show differences in hemoglobin type between these species, but were unable to determine whether the hybrids had one hemoglobin molecule with two antigenic determinants or whether they actually had two different types of hemoglobin.

INTERSPECIES HEMO-HYBRIDIZATION

The guinea fowl has been used in many species hemo-hybridization studies. These so-called 'vegetative hybridization' studies consisted of transfusions or injections of whole or fractionated blood from one species to another. Positive results in the form of the appearance of black flecks, and in some cases extensive melanin extension in the plumage of later generation chicks, have been reported from domestic fowl treated with guinea fowl blood (Penionzkevic and Misin, 1959; Stroun et al., 1963; Leroy et al., 1964, 1966; Gromov, 1970). The presence of white feathers appearing in future generations of guinea fowl treated with chicken blood from dominant white chickens has also been reported (Gromov et al., 1974, 1975). Others, however, have not been able to repeat these results (Buschinelli, 1962; Lowe and Wilson, 1965) even when using the same stock and methods (Lowe et al., 1968). This type of research is not popular at the present time, but has been completely reviewed by Styeklyenov (1978).

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Chapter 15. MUTATIONS AND MAJOR VARIANTS IN RING-NECKED PHEASANTS

Ralph G. Somes Jr.

The ring-necked pheasant has been the basis of a number of studies to try to better understand some of its traits from a practical standpoint as well as to contribute to knowledge of the genetics of the family Phasianidae. The qualitative traits that have been studied in the ring-necked pheasant can be grouped into two categories and will thus be presented in this way. One grouping deals with visible traits while the other covers biochemical characteristics. Table 15.1 summarizes the loci covered in this chapter.

FEATHER COLOR TRAITS

Nine single gene mutations that affect plumage color have been reported. Gene symbols have been assigned to only five of these loci. In this report symbols are proposed for the other four loci.

Wild-type. Coloration of the common ring-necked pheasants is assumed to be the wild-type. The male's head and neck are green with strong purplish reflection; there is a more or less complete white neck-ring; the rump is grey or yellowish-grey; the breast is bronze-red with the feathers very narrowly margined with purple-black and the ends of the feathers notched with a narrow purple-black wedge; the flank feathers are buffy tipped with blue-black. The females are a mixture of black, brown and rust dorsally; the breast and sides are rust and black. At hatching the chicks resemble Brown Leghorns. The underparts are sandy-buff; the back, head and neck are somewhat more brown, with three black stripes on the back.

Spotting or pied (*pi*). One of the first studies dealing with the inheritance of plumage color in ring-necked pheasants was reported by Phillips (1915) in which a single 'spotted' male was mated with a ring-necked female. The F_2 generation produced 68 typical ring-necked and 22 spotted chicks, and it was concluded that spotting was caused by an autosomal recessive gene. In the F_3 generation, the amount of whiteness was observed to increase. The eyes of spotted pheasants were reported to be dirty bluish-grey instead of the normal bright hazel-brown. They were also considered to be very delicate and hard to raise. No gene symbol was assigned. The symbol *pi* is now proposed for this spotting gene.

Greb (1944) reported very briefly on a recessive spotted type of pheasant that was found in low frequency in central South Dakota. As in the one reported by Phillips (1915), white markings varied considerably. In some later reviews this spotted phenotype is referred to as pied (Bruckner, 1939, 1941a; Crawford, 1974).

Melanistic, mutant or black (*M*). Bruckner studied a number of pheasant color mutants. The first one he reported was the melanistic mutant (Bruckner, 1939). Today this color type is known among fanciers as 'melanistic', 'mutant' or 'black'. Melanistic

Table 15.1. Genetic traits of the ring-necked pheasant.

Phenotype	Gene symbol	Mode of inheritance
	Morphological traits	
Pied spotting	pi, Pi^{+*}	autosomal recessive
Melanistic	M, m^+	autosomal incompletely dominant
Blue-eyed white	c, C^+	autosomal recessive
Brown-eyed white	bw, Bw^{+*}	autosomal recessive
Incomplete albinism	a, A^{+*}	sex-linked recessive
Cream	cr, Cr^{+*}	autosomal recessive
Dilute	di, Di^+	sex-linked recessive
Buff	B, b^+	autosomal incompletely dominant
Wheaten	wh, Wh^+	autosomal recessive
White neck-ring	no symbol	recessive
Wooley	wo, Wo^+	recessive
White skin	W^+	dominant
Gold	s^+	sex-linked recessive
Barring	Ba^{+*}	sex-linked dominant
Dermal melanin	id^+	sex-linked recessive
Ear-tufts	no symbol	recessive
Velvety face patch	no symbol	recessive
	Biochemical polymorphisms	
Transferrin	Tf^A, Tf^B, Tf^C, Tf^D	autosomal co-dominant
Blood group A	$Ea\text{-}A^1, Ea\text{-}A^2$	autosomal co-dominant
Blood group B	$Ea\text{-}B^1, Ea\text{-}B^2$	autosomal co-dominant
Blood group C	$Ea\text{-}C^1, Ea\text{-}C^2$	autosomal co-dominant
Prealbumin	Pa^A, Pa^B, Pa^C	autosomal co-dominant
Haptoglobin	Hp^S, Hp^F	autosomal co-dominant
G6PD	*Gpd*	autosomal
Plasma albumin	*Alb*	unknown
Hemoglobin	*Hb*	unknown
Ceruloplasmin	*Cer*	unknown
Ovalbumin	*Ov*	unknown

* Proposed gene symbols.

pheasants have apparently been around for a long time, for Bruckner (1939) quoted Dugald MacIntyre as stating that the melanistic form was first described in 1888 in England by Lord Rothschild.

The melanistic mutant is primarily black both as a chick and as an adult. The chick is predominantly black when hatched, although there is variation, with some individuals showing considerable brown; the throat, wing primaries, and abdomen are white with variation among individuals. The adult male is a brilliant black with a blue-green and purple sheen, particularly around the neck and hackle; tail feathers are barred. Females are black, but some individuals show brown barring.

Reciprocal F_1 crosses, F_2 and backcrosses with common ring-necked pheasants all showed that the melanistic phenotype is the result of a single incompletely dominant autosomal gene to which Bruckner (1939) assigned the symbol M. He was unable to distinguish between the M/M and M/m^+ genotypes in the F_2 generation, all melanistic birds of this generation being intermediate and none showing complete extension of black throughout the plumage. Other backcrosses to *M*/*M* type birds led him to conclude that a single major gene differentiates the melanistic pheasant from the ring-necked, but that maximum expression of the *M* gene depends upon modifying genes which had apparently accumulated in the melanistic as a result of selection.

There has been a controversy as to whether this trait is a true mutation or the result of selection following a cross between *Phasianus colchicus* and *P. versicolor*. Bruckner's (1939) F_1 crosses looked remarkably like pheasants of the latter species. He suggested that this gene might have mutated in Japan and spread so that it became recognized by ornithologists as a separate species *P. versicolor*. It would then at a later date have been introduced into Europe, crossed into ring-necked stock there, and selected for intensity. Others believed that it is a true mutant of *P. colchicus* and they have thus named it *P. colchicus* mut. *tenebrosus*. To this author's thinking, it would seem that in either case this is a mutation and all that is at issue here is whether it first occurred within the restricted population of the Japanese archipelago or within some other population, such as in Europe.

White mutations (*c*, *bw*, and *a*). It would appear that as many as four genetically different all-white mutations have been reported. Taibell (1928) reported on what he called an 'albino' as being the result of a single recessive gene. However, his birds all had blue eyes and thus were not technically albinos. Some time later, Bruckner (1941b) reported extensive data on a very similar white pheasant. These data conclusively showed that the blue-eyed white pheasant was the result of a single autosomal recessive gene for which he assigned the gene symbol *c*. Besides having blue eyes, these white pheasants also had pearl-white shanks and beak. This is of particular interest since similar recessive white genotypes in other species, the chicken for instance, do not have such an effect on the beak and shanks and thus it would suggest that in these two species the genes responsible are not homologous. Bruckner (1941b) also reported that sometimes the dominance of the color gene (C^+) was not always complete. An occasional white flight or small white throat patch would appear in adult heterozygotes (C^+/c) and an occasional colored feather or slight ticking would appear in homozygous whites (c/c).

Shelley (1935) reported on seven wild white pheasants in New Hampshire which had hatched from normal colored parents. The recessive nature of this white was obvious from the coloration of the parents. However, these particular white pheasants had normal brown eyes and buff-colored beaks and shanks. Similar dark-eyed recessive white birds were reported to have occurred in South Dakota in 1955, 1956 and 1957 (Morgan, 1958). It is proposed that the gene symbol *bw* be used to designate this gene.

The only reference to a true albino was made by Bruckner (1941b) in which he states, after explaining that a true albino must have pink eyes, that among the approximately million pheasant chicks hatched in nine years by the state of New York, only one true albino was found. No other information was offered on this bird. Bruckner (1969) has also reported an incomplete albinism with reduced viability, which was due to a sex-linked recessive gene. Unfortunately the only information relative to this trait consists of two sentences in an abstract. The gene symbol *a* is proposed for this sex-linked gene.

Cream, Van Buren dilute or faded (*cr*). Several mutations which dilute feather pigmentation have been reported. The one with the least information available concerning it is referred to by several names: cream, Van Buren dilute and faded. This phenotype is caused by an autosomal recessive in which males are much more affected than females (Bruckner, 1941a). Unfortunately no other information is available concerning this mutation. The gene symbol *cr* is proposed for this dilution gene.

Sex-linked dilute or blond (*di*). A second dilution factor was reported by Bruckner (1941a) in the same abstract as the cream gene. This second factor is known as blond or sex-linked dilute. Its action is similar to that of cream but the two mutants can be readily distinguished from each other in both sexes. Wentworth et al. (1967) further studied and described this mutant in a study investigating the feasibility of sexing day-old pheasant chicks by down color variations. They were able to verify its sex-linked inheritance and assigned to it the gene symbol *di*.

The effect of the blond gene on chick down color is to greatly reduce pigment to a blond color. Males still have a definite stripe on their backs, while females do not (Figure 15.1). As adults, males have the wild-type plumage while females retain the dilute blond color. Wentworth et al. (1967) suggested that the auto-sexing properties of the blond gene may be due to an interaction between embryonic sex hormones and the *di* gene, rather than to a dosage effect of the *di* gene, which would be expected to give results opposite to those observed. The *di* gene is also able to exhibit its dilution action in the presence of the melanistic gene, *M*. A new phenotype, tan, is produced when the *di* and *M* genes are both present ($di/\text{-}\ M/m^+$).

Light buff and buff (*B*). In 1957, Asmundson et al. (1964) received two phenotypically distinct color mutants from the California Department of Fish and Game. One of these was a buff in which both sexes were brown or buff with variable depth of pigmentation suggestive of the ring-necked pattern. Males had black or purplish-black feathers above the neck ring and some black in the remiges. In the second color mutant both sexes were light buff with some feathers nearly white, but with just enough pigment to be suggestive of the ring-necked pattern.

Series of crosses between these mutants and the wild-type showed that both of these phenotypes were caused by the same gene. Light buff was the homozygous expression of an incompletely dominant autosomal gene which was designated as *B*. The buff phenotype was the heterozygous expression (B/b^+). Thus buffs when mated to each other produced light buffs, buffs and wild-type in a 1:2:1 ratio. The authors also conducted studies to check for pleiotropic affects of the *B* gene on fertility, hatchability, body weight and post-hatch viability. In no instance were statistically significant differences found.

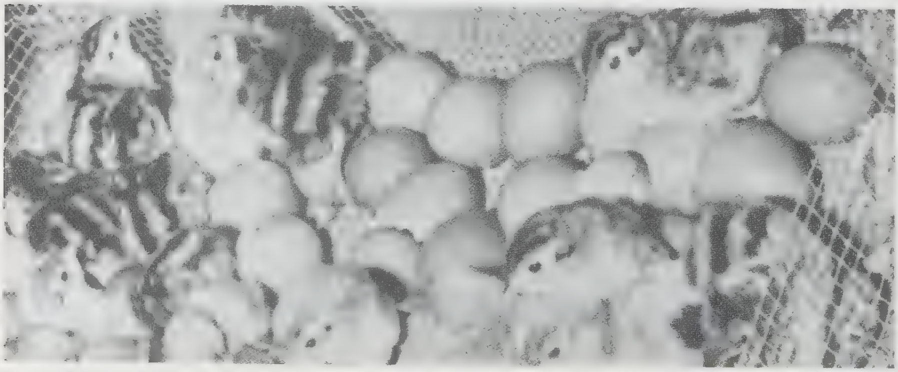

Figure 15.1. Autosexing chicks from a mating of blond male (*di/di*) x wild-type female ($Di^+/\text{-}$). Blond and wheaten chicks have a similar phenotype, but the former is sex-linked and the latter is autosomal. (Photo courtesy B.C. Wentworth).

Wheaten (*wh*). The latest plumage color gene to be reported for *P. colchicus* is the wheaten mutant (Crawford, 1974). This color was so named because of its similarity to the wheaten plumage color of chickens. The gene symbol *wh* was assigned when it was determined that the variant resulted from a single autosomal recessive gene. This mutant first appeared in 1948 on a Saskatchewan game farm.

The dilution action of the *wh* gene is greater in females than in males in the adult plumage, but as chicks both sexes have their plumage pigmentation equally reduced. In this regard the action of the *wh* gene is somewhat like that of *di*. Chicks of both sexes are a creamy white with occasional black flecking. Juveniles are white. Adult females are pale buff to creamy white, while adult males become fully pigmented. The male plumage pattern is similar to the wild-type, but the white neck-ring is absent as are the bars normally present on the tail and flight feathers; the mosaic pattern on the back is blurred and the blues and greys are replaced by a rufous coloring, so that males appear less brilliantly colored than the wild-type (Figure 15.2).

White neck-ring. A white neck-ring is not characteristic of all *P. colchicus* subspecies. Two groups, the black-necked and the Tarim or olive-rumped, completely lack neck-rings (Johnsgard, 1986). Other subspecies show a range of expression that varies from the extreme of only a few white spots in one subspecies to a very broad complete ring that is not interrupted in front in other species. All of the *mongolicus* or Kirghiz, and about two-thirds of the *torquatus* or grey-rumped groups, have well developed white neck-rings. Although no studies appear to have been made between subspecies regarding this trait, several studies have been reported that do give some insight into the inheritance of the neck-ring.

Gudelman and Cain (1978), using pedigree matings, studied ring width by comparing 16 wide-ringed sire families with families of randomly selected sires. Table 15.2 shows the

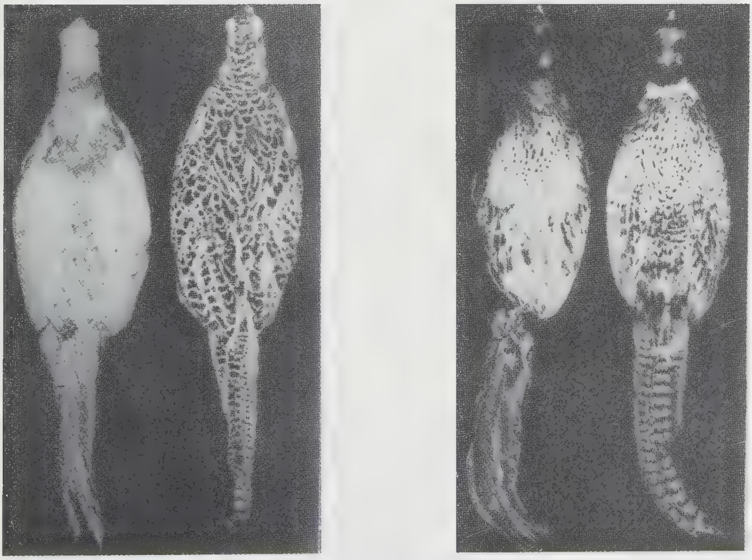

Figure 15.2. Dorsal view of wheaten and wild-type adult females (left) and adult males (right). (From Crawford in J. Hered. 65:106-107, 1974. Copyright 1974 Am. Genet. Assoc.).

Table 15.2. Genetic parameters of neck-ring width. (Adapted from Gudelman and Cain, 1978).

Parameter	Right side	Left side	Back
Correlation between sire and progeny	0.71	0.62	0.50
Heritability (estimated)	0.88	1.00	0.92
Heritability (realized)	0.88	1.00	0.82

high correlation and heritability values that they obtained. Their further characterization of the ring showed a significant correlation between ring width on the sides and the back of the neck, a high degree of bilateral symmetry, and that the number, but not the size, of the all-white and black-laced feathers in the ring was the most significant contributor to the width of the ring.

Two of the previously discussed plumage color mutants are hypostatic to the neck-ring. The recessive wheaten gene (*wh*) (Crawford, 1974), and the dominant melanistic gene (*M*) either in homozygous or heterozygous state (Bruckner, 1939), have the effect of eliminating the white neck gene.

A number of ring-necked pheasant x chicken hybrids have been described in detail (Shaklee and Knox, 1954; Malinowska and Plak, 1966; Bhatnagar et al., 1972). In each case specific mention was made of the absence of the white neck-ring, or its absence was evident from the illustrations. Thus it would appear that the white neck-ring is a recessively inherited trait. However, the wide expressitivity that is seen among the various *P. colchicus* subspecies would suggest that modifying genes have a strong influence on its actual expression.

FEATHER STRUCTURE

No mutations relating to feather structure, distribution or growth have been reported in the technical literature. For a time in the early 1980s, however, a 'wooley' ring-necked pheasant was offered for sale by an Illinois game farm. Correspondence with the game farm owner revealed that the trait was recessive. The gene may have also affected viability although reduced viability could have been due to inbreeding. Feathers sent to the author for examination were similar to those of frizzled chickens. The birds could not fly and covered pens were not needed. This gene also made the skin of homozygotes orange in color, while the skin of heterozygous carriers was a deeper yellow than was normal.

INTERGENERIC HYBRIDS

Intergeneric crosses between ring-neck pheasants and chickens, and ring-necked pheasants and turkeys show that certain well characterized genes in both the chicken and turkey appear to function also in the hybrids. By comparing the morphological characteristics of hybrids with those of the parental species it should be possible to suggest the inherited bases of some traits in each species, and to indicate where some genes of the pheasant are homologous to those in either the chicken or the turkey. Ring-necked pheasants and turkeys have white skin, and it is the wild-type (W^+) in the chicken. Crosses between yellow-skinned (w) breeds of chickens and ring-necked pheasants produced white-skinned offspring; thus it appears that the same locus with its white skin allele is present in both species (Shaklee and Knox, 1954; Bhatnagar et al., 1972). The same investigators along with Serebrovsky (1929), using hybrids produced from ring-necked pheasant males mated to female chickens, have shown that the pheasant also has three other loci, all on the sex chromosome in common with the chicken. Ring-necked pheasants have the gold (s^+), barring (B) and dermal melanin (id^+) alleles at each of their respective loci. The results from reciprocal sex-linked crosses between ring-necked pheasants (gold barred) and Columbian Plymouth Rock chickens (silver nonbarred) showed segregation to be as expected in a chicken x chicken mating with these two sex-linked genes (Bhatnagar et al., 1972). Other color genes, such as dominant white (I) in the chicken (Danforth and Sandnes, 1939; Asmundson and Lorenz, 1957), and dominant slate (D), black (B), sex-linked brown (e) and sex-linked albinism (n^a) in the turkey (Asmundson, and Lorenz, 1957) all express themselves in the hybrid and even segregate as would be expected.

With turkey x ring-necked pheasant hybrids, all the characteristic head and breast features of the turkey (snood, median wattle, caruncles and beard) are absent and the head is completely feathered except around the eyes (Asmundson and Lorenz, 1957). With chicken x ring-necked pheasant hybrids the comb, wattles, and earlobes of the chicken are similarly absent (Shaklee and Knox, 1954; Asmundson and Lorenz, 1957; Malinowska and Plak, 1966) as are the neck-ring, ear-tufts and velvety red feathering on either side of the head of male ring-necked pheasants (Shaklee and Knox, 1954). In hybrids with both turkeys and chickens, leg spurs are absent or very small (Shaklee and Knox, 1954; Asmundson and Lorenz, 1957; Malinowska and Plak, 1966). Since well characterized genes of the turkey and chicken express themselves in the hybrid, it might be expected that other less well characterized traits would do likewise. The neck-ring, ear-tufts and velvety face patch genes of the ring-necked pheasant are probably recessive, while those that result in a fully feathered head without the head features of either the chicken or the turkey are dominant.

BIOCHEMICAL POLYMORPHISMS

Several studies have been undertaken to look for polymorphisms among blood, tissue and egg proteins of the ring-necked pheasant. Variability has been found among some of these proteins, but only a few have been shown to be definitely genetically controlled.

Transferrin-conalbumin (Tf^A, Tf^B, Tf^C, Tf^D). One of the most studied of the biochemical traits has been the variations that are evident among the iron-binding proteins of serum (transferrin) and egg white (conalbumin). Baker et al. (1966) found that both egg white conalbumin and serum transferrin consisted of two types, a 'fast' band and a 'slow' band, when examined electrophoretically. Ogden et al. (1962), when examining these two proteins in the domestic fowl, suggested that the observed parallel variation was probably due to both proteins being under the genetic control of a single locus. It would appear that the same control is working in the ring-necked pheasant, as suggested by Baker et al. (1966). Their ring-necked pheasants showed 'fast' bands only, 'slow' bands only, or both a 'slow' and a 'fast' band. They proposed that the gene symbol Tf be used to designate both transferrin and conalbumin with Tf^S referring to the 'slow' variant and Tf^F referring to the 'fast' variant, indicating that a pair of codominant genes at a single locus were controlling this observed variability.

Four homozygous transferrin types were found in a study designed to explore the use of transferrin polymorphisms in field studies of geographically isolated populations of wild ring-necked pheasants in Iowa (Vohs and Carr, 1969). By means of reciprocal matings, it was determined that a single autosomal locus consisting of four codominant alleles was involved. The nomenclature proposed by Baker et al. (1966) was not suitable for designating these new findings and thus the alleles were redesignated *A-D*. Under this designation Tf^A and Tf^D were the new alleles and Tf^B and Tf^C were the same as Tf^F and Tf^S respectively. A somewhat similar study was conducted using ring-necked pheasants from both South Dakota and Pennsylvania (Montag and Dahlgren, 1973). These researchers identified the *A-C* alleles but did not find the *D* allele. Through breeding tests they verified the findings of Vohs and Carr (1969). Lucotte and Kaminski (1976) have shown that the Tf^B/Tf^C type of egg white conalbumin has a stronger inhibitory effect on the growth of *Saccharomyces cerevisiae* than does the more common Tf^C/Tf^C type. They interpret this as a possible example of heterosis at the molecular level which may account for the maintenance of polymorphism for this protein.

Blood groups (*Ea-A, Ea-B, Ea-C*). Blood group factors were studied in wild ring-necked pheasants in Iowa (Vohs, 1966) by means of agglutination reactions of red blood cells with typing fluids. Three factors, A, B and C, were identified. Test matings for each of these factors showed that they segregated in a Mendelian fashion. Each factor seems to be inherited as a dominant or codominant to an as yet undetected recessive or codominant factor. Factors A and C, and B and C were shown to be independent of each other, and thus it was assumed that A and B were also independent of each other. Three separate blood groups factors *Ea-A*, *Ea-B* and *Ea-C*, each with at least two alleles, were proposed to explain these findings.

Prealbumin (Pa^A, Pa^B, Pa^C). Using a discontinuous buffer, Baker et al. (1966) were able to show the presence of three main bands in the serum prealbumin region II. No more than two of these bands appeared in any one bird and all six possible combinations were observed among a sampling of ten populations consisting of 64 birds. It was proposed that these variants were controlled by three codominant alleles at a single locus, Pa^A, Pa^B and Pa^C.

Haptoglobin (Hp^S, Hp^F). Using an 8-slot borate starch-gel system for best resolution, polymorphisms have been detected for serum haptoglobin (Baker et al., 1966). Among 28 birds examined, three haptoglobin types were identified. Birds had either 'slow' band only, 'fast' band only or both 'slow' and 'fast' bands. Although no breeding tests were made, the occurrence of these three types of haptoglobins suggested genetic polymorphism controlled by two codominant alleles at a single locus, Hp^S and Hp^F.

Glucose 6-phosphate dehydrogenase (G6PD). Bhatnagar (1969) studied erythrocyte G6PD isozyme patterns for chickens, ring-necked pheasants, and their reciprocal hybrids by means of starch-gel electrophoresis. The pheasant G6PD isozyme bands were different from those of the chicken. Thus when both the male and female hybrids from the reciprocal crosses had similar isozyme patterns representing both of their parental types, it was assumed that the genes controlling the production of G6PD in both chicken and pheasant erythrocytes were located on an autosome. Baker et al. (1966), in an appendix to their paper and with no data presented, mention that they had found polymorphism of G6PD in an English population of *P. colchicus*.

Plasma albumin. Sarvella and Morris (1976) using disc electrophoresis studied plasma albumin bands in four species and their F_1 hybrids. Hybrids of ring-necked pheasants with both Japanese quail and turkeys had only one band, while hybrids between pheasants and chickens had two bands. Thus the ring-necked pheasant would appear to have the same plasma albumin allele as that of both the Japanese quail and the turkey, but different from that of the chickens used in their study. Baker et al. (1966) reported that one of their ring-necked pheasants had an albumin pattern that was markedly different from that of the other 146 individuals examined. However, they did not examine this difference further or make any crosses.

Hemoglobin. Hemoglobin variability was looked for among six mutant strains of chickens, junglefowl, ring-necked pheasants, and their F_1 hybrids by Brush (1967). He was unable to detect any polymorphisms for either the major or minor hemoglobin components in any of the stocks examined. However Baker et al. (1966) reported that one of their 129 pheasants had a markedly abnormal hemoglobin pattern. The major hemoglobin component was reduced in quantity from its normal 80 percent to ten percent of the total hemoglobin, while there was more than the usual amount of the minor component. It was suggested that this pheasant might be suffering from a genetic defect in the production of a specific hemoglobin polypeptide chain. This variant was not studied further.

Ceruloplasmin. In a small group of eight pheasants from Illinois, Baker et al. (1966) found one bird that had a 'slow' ceruloplasmin band while three others had 'fast' bands. Four others had bands with electrophoretic mobilities that were intermediate to the 'fast' and 'slow'. Although no further studies were undertaken, these observations suggested the presence of polymorphism in this serum protein.

Ovalbumin. Electrophoretic patterns of pheasant egg-white ovalbumin showed it to have three forms, similar to those of the chicken (Baker et al., 1966). The A_1 form had two phosphate residues/mole, A_2 had one, and A_3 had no phosphate. One pheasant out of 88 had a very unusual pattern with four bands present. They also reported that some of their English *P. colchicus* populations contained a polymorphism, but not the same one as just mentioned for their American sample.

Oncogenes (*c-myb, c-myc, c-src*). Pheasant genomic DNA from erythrocytes has been probed with proviral sequences from avian myoblastosis virus (*myb*), avian myelocytosis virus (*myc*), and avian sarcoma virus (*src*). All three oncogene probes detected specific genomic sequences (Andersen et al., 1985). Hybridization was under such stringent conditions that nearly complete homology of sequences was necessary for detection to occur. Thus pheasants would appear to have the oncogenes *c-myb, c-myc* and *c-src* in common with the domestic fowl.

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Chapter 16. MUTATIONS AND MAJOR VARIANTS IN DOMESTIC DUCKS

F.M. Lancaster

There seems little doubt that the ancestor of all domestic ducks is the common mallard (*Anas platyrhynchos platyrhyncos*). In accordance with current practice (Jaap and Hollander, 1954), traditional gene designations will be amended in this review to reflect more clearly relationship to the wild-type. Variations described in this chapter mostly involve color and morphology. Only a few variants in other body systems of domestic ducks are known; access to the literature on these may be obtained from other chapters. Knowledge concerning gene homology between domestic and muscovy ducks is reviewed in Chapter 17.

PLUMAGE COLOR VARIANTS

Minor variations in the wild-type pattern are responsible for the characteristic appearance of several breeds. These variations are explained by two distinct multiple allelic series. One and perhaps two mutations cause extension of black. There are three genetic kinds of color dilution, and several factors causing white plumage or markings.

Wild-type or mallard pattern. In the day-old mallard duckling the dorsal surface is a dark olivaceous black. On the back of the duckling are four yellow spots which form the corner points of a rectangle situated anteriorly just behind the wings and posteriorly just in front of the tail. The sides of the head are yellow with one or two dark ocular stripes extending from the base of the bill to the dark dorsal area on the back of the head. The ventral surfaces of the throat and wings are yellow. The remainder of the ventral surface is dull yellow which has a muddy black undercolor extending out to the surface in parts giving a mottled appearance.

Adult male. Head and neck are rich iridescent green-black divided from the claret-colored breast by a white neck-ring. Back and rump are rich green-black running back from between the shoulders. Flank, side and belly are blue-grey finely pencilled with black. The brilliant blue-green iridescent speculum is edged with two black and white bands on each wing. Ventral wing surfaces are white. Dorsal surface of wings and primary feathers are brownish grey.

Adult female. Head is rich buff-brown finely streaked with brownish black markings. The dark markings meet in certain areas to form a well-defined pattern. There is a dark line running from the base of the bill back through the eye and a dark patch on the crown which runs down the back of the neck. Wing specula are brilliant as in the male. Ventral wing surfaces are white. The remainder of the plumage is a rich brown, each feather distinctly pencilled with black or very dark brown.

Restricted, mallard, dusky series (M^R, M^+, m^d). This triple allelic series was first described by Jaap (1934). It is autosomal and there is complete dominance among members of the series in the following order: restricted (M^R)>mallard (M^+)>dusky (m^d).

Mallard (M^+) in this series is the wild-type described above. It is not possible to give detailed descriptions of restricted (M^R) and dusky (m^d) variants due to limitations of space. They are adequately covered in the papers of Jaap (1934) and Lancaster (1963). Basically the dusky pattern is darker and plainer than the mallard both in the day-old and adult while the restricted pattern is lighter in the day-old and has white on the wing fronts at maturity. The main differences are summarized in Tables 16.1 and 16.2.

Recently, Campbell et al. (1984) provided evidence of a fourth member of this series. The new gene, symbolized by M^B, is responsible for completely black plumage and has been postulated to be the true top dominant member of the series. The authors consider this type of black to be different from extended black (*E*) as described by Jaap (1934) and Lancaster (1963). However, they did not use a known source of extended black (*E*) in test matings and interpretation of evidence to support their view needs further proof. The presence of white breast markings in their birds suggest that M^B may in fact be identical with *E*. This aspect is discussed further in the section on white spotting.

Dark phase, light phase, harlequin phase series (Li^+, *li*, li^h). The dark phase (Li^+) and light phase (*li*) pair of autosomal genes was first reported by Jaap (1933b). The

Table 16.1. Appearance at day-old of the restricted, mallard, dusky series. (Modified from Lancaster, 1963).

Type	Dorsal spots	Yellow face and ocular head stripes	Dark down pattern	Ventral wing surfaces	Breed examples
Restricted (M^R)	obscured by surface yellow	obscured	restricted to head and tail	yellow	Silver Appleyard, Pekin (hypostatic)
Mallard (M^+)	present	present	fully extended	yellow	Rouen, Brown (Grey) Call
Dusky (m^d)	absent	absent	fully extended	darkly pigmented	Khaki Campbell, Buff Orpington

Table 16.2. Adult appearance of the restricted, mallard, dusky series. (Modified from Lancaster, 1963).

Type	Ventral wing surface	Ocular head stripe (females)	Dorsal surface of wing front	Wing speculum	Claret breast and white neck-ring (males)
Restricted (M^R)	white	present	white	present	present
Mallard (M^+)	white	present	pigmented	present	present
Dusky (m^d)	pigmented	absent	pigmented	absent	absent

series was later extended by Lancaster (1963) with the addition of harlequin phase (li^h). The order of dominance is $Li^+ > li > li^h$ and dominance is complete. For simplicity it is assumed that the mallard (M^+) allele of the previous series accompanies all three phases in the present discussion. Interactions between the two series are fully documented by Jaap (1933b) and Lancaster (1963).

Dark phase (Li^+). This is the wild-type gene present in the mallard and the Rouen breed. It allows full expression of any of the three M^+ alleles which happen to be present.

Light phase (li) and *harlequin phase* (li^h). As in the previous series full descriptions of light and harlequin phases are not possible but can be found in the references cited above. The main effect of the two alleles is a progressive lightening of the plumage color and replacement of many of the colored feathers by white ones. The only exception to this is the claret-colored breast of the male which is actually extended in li and li^h. The changes to the wild pattern wrought by li and li^h are similar but in the case of li^h the effects are exaggerated. Tables 16.3 and 16.4 summarize the main differences.

Extended black (E). Fully extended black in the duck is autosomal dominant to nonextended black (Phillips, 1915; Jaap and Milby, 1944). When homozygous the recessive gene e^+ allows full expression of the wild-type. In contrast E causes solid black

Table 16.3. Day-old characteristics of Li^+, li and li^h. (Modified from Lancaster, 1963).

Type	Dorsal body surface	Ventral body surface	Dorsal spots	Ocular stripes	Breed examples
Dark phase (Li^+)	darkly pigmented	pigmented	normal	usually two	Rouen, Brown (Grey) Call
Light phase (li)	darkly pigmented	yellow	elongated	one	Silver Appleyard
Harlequin phase (li^h)	mainly yellow	yellow	obscured by yellow	obscured by yellow	Welsh Harlequin

Table 16.4. Expression of Li^+, li and li^h at maturity. (Modified from Lancaster, 1963).

Type	Distribution of claret on breast of male	Extension of body pigment (grey in male, brown in female)	Dorsal wing surface	Ventral body surface (females)	White neck-ring (males)
Dark Phase (Li^+)	normal (breast only)	normal	pigmented	pigmented	normal
Light phase (li)	extended on to shoulders, sides and breast	partially replaced by white	pigmented	white	enlarged
Harlequin phase (li^h)	further extended in above areas	mainly replaced by white	mainly white	white	enlarged

pigment to be laid down in all areas except those affected by genes for white spotting and is completely epistatic to all genes at the *M* and *Li* loci. The gene is found typically in the Black Orpington, Black Cayuga and Black East Indian.

Hawes (1965) has demonstrated that clubbed down accompanies extended black, as it does in chickens and turkeys, probably as a pleiotropic effect. He examined 24-day-old embryos and found clubbing to be expressed as a shortening and buckling of the plumules over the back, giving a matted appearance to the down. Morphology of juvenile and adult feathers was not adversely affected by presence of extended black (*E*).

There are three known dilution genes in the duck, *Bl*, *d* and *bu*. When present singly or together in various combinations against the alternative background patterns of *E* or e^+, they are responsible for a large number of color and pattern effects in the duck. Several of these are summarized in Table 16.5.

Blue dilution (*Bl*). The mode of inheritance of blue dilution in the duck has been known for many years (Wright, 1902; Lamon and Slocum, 1922; Ghigi and Taibel, 1927; Jaap and Milby, 1944). The gene is incompletely dominant and autosomal and its main effect is to dilute black pigment to blue-grey in heterozygotes. In homozygotes the birds are much paler and in some cases almost white. Because *Bl* is primarily a diluter of eumelanin its effect will differ according to whether it is associated with *E* or e^+ (Table 16.5).

Brown dilution (*d*). Brown dilution in the duck was first investigated by Punnett (1930, 1932) and by Walther et al. (1932) who independently concluded that the gene responsible (*d*) was recessive and sex-linked. Like blue dilution it exerts its main effect on black pigment changing it to a dark chocolate brown. Brown dilution has little effect on pheomelanin in the plumage but it obscures the brilliant wing speculum (Table 16.5).

Blue and brown dilutions together act additively giving an even paler type of bird as indicated in Table 16.5.

Table 16.5. Separate and combined effects of blue and brown dilutions on *E* or e^+ plumage types.

Dilution genotypes	Combined with E/E	Breed examples	Combined with e^+/e^+	Breed examples
$Bl/bl^+ \; D^+/D^+$	blue-grey	Blue Swedish, Blue Orpington	black areas of wild-type diluted to blue-grey	Apricot Call (?), Saxony (?)
$Bl/Bl \; D^+/D^+$	blue-splashed white	light segregates from above	similar to above but paler	Apricot Call (?), Saxony (?)
$bl^+/bl^+ \; d/d$	uniform chocolate	Chocolate Orpington, Chocolate Indian Runner	black areas of wild-type diluted to chocolate brown	Fawn Indian Runner, Khaki Campbell
$Bl/bl^+ \; d/d$	lilac (light blue with pinkish shade)	none	buff	Buff Orpington
$Bl/Bl \; d/d$	lilac-splashed white	none	pale buff	light segregants from above

Buff dilution (*bu*). Buff dilution in ducks was first reported by Lancaster (1963). Reciprocal matings between the Buff Orpington and the Khaki Campbell demonstrated the presence of a further sex-linked recessive dilution gene which was named buff dilution (*bu*). When pale Buff Orpington males ($Bl/Bl\ d/d\ bu/bu$) were crossed with Khaki Campbell females ($bl^+/bl^+\ d/\text{-}\ Bu^+/\text{-}$) the male progeny at day-old had darker down color and darker bill, legs and feet than the females. At maturity these differences were still present.

Recessive white (*c*). White plumage in all the common white breeds of domestic ducks is caused by an autosomal recessive gene (Jaap, 1933a; Mazing, 1933). When the recessive white gene is homozygous it is completely epistatic to all other genes affecting plumage color and pattern.

Snow-white down. The down color of recessive white ducklings is normally yellow but Krizenecky (1961) described a mutation which changed the yellow down to snow-white in the day-old. Although the gene responsible was established as being recessive, its autosomal or sex-linked status was not determined.

Complementary white. McIlhenny (1941) reported on the mating of two white drakes with four white ducks originating from a flock of wild mallards. All the progeny from these birds were colored indicating complementation between two different kinds of recessive white. More recently Lancaster (1963) tried to repeat these results by mating all the white breeds available in all possible combinations but was unable to detect any complementary effects.

Albinism. According to Scott (1957) pure albinism has not been recorded in waterfowl. There is a painting of an albino mallard female by T.M. Shortt in Kortright (1942) but no reference to the source of information is given.

Dominant bib (*S*). This is a white marking of the neck and upper breast region on a colored background. It is extremely variable in size and often extends up to the base of the bill and beyond. Typically this pattern can be seen in the Blue Swedish and Blue Orpington breeds. Several workers have investigated its inheritance. Jaap and Hollander (1954) suggested it might be an interaction effect. Lancaster (1963) found that the gene responsible behaved as an autosomal dominant and appeared to be very closely linked with extended black (*E*). It showed variable expressivity and in some breeds like the Cayuga and Black East Indian it lacked penetrance. In the breeds examined by Lancaster (1963) the two loci were linked in coupling phase and test matings failed to recover any recombinants. However, Robinson (1924) described and presented a photograph of Landsmeer ducks from Holland which have a similar plumage pattern to the Rouen (e^+) but appear to display well developed dominant bibs (*S*). Recently, Roberts and Roberts (1986) published photographs of the Hook-billed breed which also seems to have the same repulsion phase combination ($e^+/e^+\ S/S$).

Lancaster (1963) found evidence of latent or nonpenetrant bibs in Cayuga and Black East Indian ducks when they were crossed with Khaki Campbells. Robinson (1924) mentioned that most of the early Cayugas in the last century had white markings and that these had since been removed by selection.

The discussion on dominant bib is relevant to the elucidation of the M^B gene mentioned earlier. Photographs of ducks carrying M^B by Campbell et al. (1984) appear to show well-developed bibs. Appropriate matings of these birds with extended black (*E*) and nonextended black (e^+) birds should establish whether or not M^B and E are the same gene.

Recessive bib (*b*). Another type of bib was reported by Jaap (1933a) in pure mallard stock. The white area was large and shield-shaped and occupied most of the claret breast region of the male and a similar area in the female. Jaap stated that the outline of the bib was different from that of the Blue Swedish. The furthest forward it extended was the neck-ring region. It did not extend upward under the chin as in some Orpington and Swedish ducks. In contrast to dominant bib the shape and size of recessive bib was remarkably constant. Jaap found that this type of bib was completely recessive and autosomal.

White primaries (*w*). One or more white primaries in ducks can result from the presence of one or both of the genes for white primaries (*w*) and runner (*R*). Since the latter also produces white in other areas it will be examined separately. The white primary gene (*w*) was first described by Jaap (1933a) who found it was autosomal and recessive. In Jaap's (1933a) experiments white primary ducklings were obtained from wild mallards. This pattern can also be seen as a breed characteristic in the Blue Swedish. Jaap (1933a) found that at day-old all the extremities of ducklings with white primaries (bill, feet and wing tips) had pale yellow markings and that the extent of these markings was correlated with the number of white primaries.

Runner pattern (*R*). The runner pattern is a complicated arrangement of white markings on a colored background. It is found typically in the Fawn and White Indian Runner. The pattern has white markings in three main areas:

- upper neck region with narrow projections from the neck to the eye and up behind the bill;
- on the ventral surface of the lower abdomen;
- on the wings (primaries, secondaries, and wing bow).

There is considerable variation in size of these white areas due to modifying genes.

Jaap (1933a) found that the runner pattern was due to an incompletely dominant autosomal gene (*R*). He showed that in the absence of the gene for white primaries (*w*) the runner gene, *R*, was also capable of producing white primary feathers when in the heterozygous state. Jaap (1933a) found some depigmentation in the bill, legs, and feet of heterozygous runner ducklings but in homozygotes these areas were always yellow. Lancaster (1963) observed that modifying genes can also influence the expression of *R* in its heterozygous form and included a broad white neck-ring in both sexes as a further manifestation of Rr^+.

Lancaster (1963) found that the presence of the runner gene (*R*) could be detected in white breed ducklings (c/c) at day-old. Ducklings carrying the runner gene (R/R or R/r^+) exhibited a ghost pattern where white down on a yellow background followed the runner configuration.

Magpie pattern. Lancaster (1963) showed that the magpie pattern results when dominant bib (*S*) and the homozygous runner pattern are brought together. This can be found in the British Magpie breed which has the genotype E/E S/S R/R. In the magpie pattern the head and neck are white except for a black cap on the top of the head. The back is solid black from between the wings at the front to just below the tail at the rear. The whole of the wings, breast, and abdomen are pure white.

INHERITANCE OF OTHER TRAITS

White skin and bill (*Y*). In breeds like the Pekin and White Campbell the presence of xanthophylls in the diet causes yellow pigment to be laid down in the bill and skin. The Aylesbury, however, possesses a gene which prevents the deposition of yellow pigment in these areas. The Aylesbury has orange shanks and feet but the rest of the skin and bill are pink or white.

Rendel (1940) reported that the gene responsible for white bill and skin was dominant and autosomal. Since no symbols were presented in Rendel's paper it is suggested that Y and y^+ be used. The presence or absence of dark melanic pigment in the bill and legs of some breeds is due to the action of plumage color genes such as C^+, R and E. Some of these genes are completely epistatic to the Y locus in these areas.

Hook-billed variant. Darwin (1896) described and illustrated the Hook-billed breed which was characterized by a downward curving beak. It was regarded as an ancient breed, having been reported as early as 1676. An informant advised Darwin that when hook-billed ducks were crossed with normal ones, many of the progeny had an upper mandible shorter than the lower. Subsequent writers made no mention of the variant and it has been presumed to have been lost. However, Roberts and Roberts (1986) have now published recent photographs of the breed. Their stock came from Holland, where it existed long before the time of Darwin and where it is still kept (Roberts and Roberts, 1988, personal communication). Genetics of the trait has not been investigated.

Egg shell color. The wild mallard lays a light buffish-green egg. Most domestic ducks produce eggs with either greenish-blue or white shells. Mazing (1933) found that the gene for white shell color was recessive but the abstract of his work does not specify whether it is sex-linked or autosomal. Since no symbols were presented in the report, G^+ is suggested for the dominant green egg shell gene of the wild-type.

In the Black East Indian and in some strains of Cayuga the first egg laid is often jet black but turns to dark grey and then light grey as successive eggs appear. This effect seems to be due to the extended black gene (E) whose influence carries over into the shell gland. Interaction of E and G loci has not been investigated.

Crest (*Cr*). The Crested breed has several color varieties which all have a raised crest of skin and feathers towards the back of the head. Studies by Rüst (1932) showed that crested ducks are heterozygous for an incompletely dominant autosomal gene. When the gene is homozygous it is completely lethal and the embryos fail to hatch. They often suffer from exencephaly, having parts missing from the cranium, and the bill is abnormal. The gene responsible exhibits variable expressivity in both heterozygotes and homozygotes. In some of the heterozygotes the gene is not completely penetrant and the birds appear normal. Cr is suggested as an appropriate symbol for this gene.

Micromelia (*mi*). Ash (1966) described a micromelic lethal condition in White Pekin embryos. Body size was reduced, the extremities were conspicuously shortened, and feathering was retarded and abnormal. Further study (Kelly and Ash, 1976) of down feathers revealed many abnormalities suggesting a common source in defective embryonic mesoderm. Most of the affected embryos survived until final stages of incubation but none was able to hatch. The condition was found to be autosomal and recessive. No gene symbol was assigned to it. The symbol mi is now proposed.

Hereditary tremor. A hereditary tremor condition was observed in domestic ducks in Sweden (Dyrendahl, 1958). There were indications that it was caused by a single autosomal recessive gene, but adequate proof was never obtained. Tremors of the whole body were evident at hatching. Most affected ducklings died from water and feed starvation, but a few survived. They varied in severity of tremors. The condition was reproduced in matings of defective male with defective and normal females, but numbers of observations were inadequate for genetic analysis.

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Chapter 17. MUTATIONS AND MAJOR VARIANTS IN MUSCOVY DUCKS

C. Fedeli Avanzi and R.D. Crawford

Ten plumage color mutations have been studied genetically in the muscovy duck (Table 17.1). Alone and in combination, these ten create a large array of color phenotypes. Other variants are known to breeders and aviculturists but genetic explanation of them has not been adequately documented. There are no known genetic variants affecting morphology, and there has been only very limited study of blood and biochemical traits.

Muscovy duck genetics has been hampered by the reluctance of some investigators to adopt modern genetic nomenclature. This nomenclature requires acceptance of a wild-type as standard (Jaap and Hollander, 1954). Origin and ancestry of muscovy ducks is not in doubt, and general description of the wild ancestor is well documented (see Chapter 1). Arguments persist about black and brown in the wild plumage, and about effects of the environment on these colors; they should be resolved by further study. In this chapter modern nomenclature is purposely adopted to encourage that further study.

The ability of muscovy ducks to hybridize with other anseriform genera and species is well known (Gray, 1958). The most important hybrids to the poultry industry are those between muscovy and domestic ducks. Large numbers are produced for meat in some countries. Reciprocal crosses yield quite different results (Gray, 1958). In progeny from domestic drake and muscovy duck, the females are smaller than males, they have no crest, and they have good egg production but the eggs are small and incapable of fertilization; the hybrid males are large, they have strong libido, and they occasionally are fertile. The reciprocal cross, muscovy drake x domestic duck, is the mating used for commercial production. Male and female hybrids are similar in size and conformation, and both are crested. Females do not develop sexually. Males develop some sexual behavior but they usually are sterile. The feasibility of hybridization between muscovy and domestic ducks has given rare opportunity for study of comparative genetics, to determine whether similar variants in the two species are due to the same mutations and/or to mutations at the same loci. Knowledge of several muscovy duck variants has been extended in this way. There is opportunity for much more study of comparative genetics in the two species from observation and measurement of the commercial hybrids.

PLUMAGE COLOR VARIANTS

Wild-type. Plumage of the wild muscovy duck (*Cairina moschata*) is presumed to be identical to the black phenotype in domestic birds. Phillips (1922) has provided the most detailed description, and that by Delacour (1959) is similar. Ducklings are seal-brown above with a yellow spot on each wing and on each side of the rump; the underside is yellow; there is a dark line behind the eye; face and forehead are yellow. Except for differences in eye markings, muscovy ducklings are very similar to mallards (*Anas platyrhynchos*). Immature birds are uniformly brown-black with little gloss. It has not yet been resolved whether the brown tone is real or an artifact of weathering. Adults of both sexes are also uniformly brown-black but with a metallic sheen of purple and green. In domestic birds, new feathers are jet black and they can fade to brown before they are molted;

Table 17.1. Plumage color variants in muscovy ducks.

Trait	Gene symbol	Mode of inheritance
Atipico, dusky	a, A^+	autosomal recessive
Barring	b, B^+	autosomal recessive
Brown-rippled	br, Br^+	autosomal recessive
Chocolate	ch, Ch^+	sex-linked recessive
Blue dilution	N, n^+	autosomal inc. dominant
Lavender	l, L^+	autosomal recessive
Sepia, faiogeno	f, F^+	autosomal recessive
Self-white	P, p^+	autosomal inc. dominant
Duclair piebald	d, D^+	autosomal recessive
White head, canizie	C, c^+	autosomal dominant

the same may pertain in wild birds but it has not been adequately documented. In both sexes a prominent white patch develops in the wings with advancing age, involving under wing coverts, upper wing coverts, and axillaries. Development and spread of the white patch should be studied further in both wild and domestic birds.

Atipico, dusky (a). One variant is known in muscovy ducks which extends dark pigmentation. It is expressed primarily in duckling down, with little effect on juvenile and adult plumages. Taibel (1956) described the phenotype, called atipico, in which wild-type pattern is totally replaced by seal-brown. The condition was autosomal recessive and the gene symbol a was assigned. Hollander and Miller (1965) studied the same or a very similar mutant which they have called dusky, to conform with terminology for the homologous variant in domestic ducks. Theirs differed phenotypically from Taibel's in having a light bib.

A dusky domestic drake was mated to several muscovy ducks, including one that was atipico, producing hybrid offspring that were dusky-atipico, thus providing proof that the mutations in the two parent species were homologous (Hollander and Miller, 1965). However gene expression in the two species may be different. The dusky mutation in domestic ducks causes extensive darkening in down, juvenile, and adult plumages (see Chapter 16), but in muscovy ducks atipico is readily apparent only in duckling down. Careful examination will reveal that a/a juveniles and adults are darker under the wings than A^+/A^+ and A^+/a individuals which are otherwise uniformly black.

A very similar variant, also called atipico, was found by Colonna-Cesari (1973). His ducklings had a yellow bib and very shiny black bills and feet. The bib persisted as a white patch in juveniles and adults. His interpretation was that this atipico was autosomal dominant and that it formed a multiple allelic series comprising atipico (B), wild-type (b^+), and barred (b). It has been suggested by Hoffmann (1988, personal communication) that this atipico would more accurately be called dominant black. Further confirmation of this variant and its alleles is needed.

Barring (b). Taibel (1954a) reported a variant in which transverse stripes or bars occur on otherwise pigmented feathers. The condition appeared to be autosomal and recessive, and it was assigned the gene symbol b. It is expressed in both down and juvenile plumages. Ducklings have yellow down with black flecks, and down feathers of the tail are tipped with black; the bill is leaden color rather than black as in wild-type or flesh color as in true white ducklings (Hoffmann, 1988, personal communication). Juvenile feathers have the barring pattern, accompanied by pale undercolor which is advantageous in commercial production. The pattern disappears in adult birds. Barring is clearly evident in black individuals but it can also be identified in those with blue dilution (Fedeli Avanzi, 1970).

Brown-rippled (*br*). A new mutant was described by Hollander (1968) who named it brown-rippled and assigned the gene symbol *br*. It was shown to be autosomal recessive and distinct from several well-known variants. Ducklings had brown down feathers and their beaks and feet were brown. A barring pattern was evident in juveniles and adults which otherwise had feathers with grey background color and sepia bars. There was no sex difference in barring pattern. In testcrosses, Hollander (1968) found brown-rippled to be expressed in the presence of lavender, atipico, and piebald. He also produced birds that were both barred and brown-rippled (*b/b br/br*), expressing both kinds of barring in the same individual.

Chocolate (*ch*). In many species of birds and mammals there has been a mutation affecting eumelanin production resulting in brown rather than black pigment. Pheomelanin production is usually not altered so that red pigments are unchanged. The khaki mutation (*d*) in domestic ducks is of this sort (see Chapter 16).

A brown variant in muscovy ducks is known in many countries. It was investigated by Hollander (1970) who named it chocolate. He did not assign a gene symbol, but *ch* is now popular usage. He found that the condition was sex-linked and recessive. In ducklings, juveniles, and adults all black pigment is replaced by brown, and reddish (pheomelanin) pigment is unchanged.

The khaki mutation (*d*) of domestic ducks is also sex-linked recessive and has phenotypic effects like those of chocolate in muscovy ducks. Hollander (1970) produced reciprocal hybrids between chocolate muscovy and Khaki Campbell and showed that khaki and chocolate are homologues or alleles.

In domestic ducks, the khaki gene can combine with others to produce various shades of buff. Buff muscovy ducks have been produced by combining chocolate (*ch*) and brown-rippled (*br*), and it has been speculated that very light buff or pastel could result from combining *ch* and the lavender mutation (Hollander, 1988, personal communication.).

Blue dilution (*N*). Three mutations in muscovy ducks can be classed as dilutions: blue dilution, lavender, and sepia.

Blue dilution in muscovy ducks was described by Taibel (1954b) as being a heterozygous condition similar to that in Blue Andalusian chickens and Blue Swedish ducks (Figure 17.1). One homozygote is black, the other is pearl-grey, and the heterozygote is blue or slate-pearl-grey. Taibel (1954b) used the gene symbol *N* (nero) and designated the black phenotype as *N/N*, blue as *N/n*, and pearl-grey as *n/n*. It would be appropriate now to reverse designations of homozygotes to indicate that the mutation is incompletely dominant autosomal *N* and the wild-type is recessive n^+, to conform with nomenclature for similar conditions in other poultry species.

Heterozygous ducklings have dark green-grey down and the four yellow spots on the dorsum are clearly evident. Adults are dark blue-grey; bill and feet colors are diluted. Pearl-grey ducklings are isabella-yellow. Adults have pale lilac plumage which is darker in the neck and tail, with pale bill and feet.

Although this mutation has similar action to that in domestic ducks, it is evident that the two are not homologous. Lancaster (1963) made reciprocal matings of blue and black Orpingtons with blue and black muscovy ducks. His data indicated that blue hybrids arose only in those matings where the blue mutation had been contributed by the *Anas* parent. Hence the two mutations are at different loci since *Anas* blue is epistatic and *Cairina* blue is hypostatic in hybrid combination. But verification is needed since the blue muscovy ducks used in these crosses may have been lavender (*l*) and not nero (*N*) (Hollander, 1988, personal communication).

Lavender (*l*). A true-breeding blue phenotype in muscovy ducks was described by Hollander and Walther (1962), who named it lavender and assigned the gene symbol *l*. Ducklings retain the wild-type pattern but seal-brown areas are diluted to pale grey. Adults are dull grey in color without any sheen to the feathers. This phenotype is similar to that of lavender in chickens (see Chapter 5) in which both eumelanin and pheomelanin are diluted. Hollander and Walther (1962) showed that lavender in muscovy ducks is autosomal recessive, and that is it not allelic to barring (*b*) and atipico (*a*).

Sepia, faiogeno (*f*). A peculiar color dilution was described by Taibel (1961) who named it faiogeno (sepia) and assigned the gene symbol *f*. It would be of interest to determine the biochemical nature of its gene action. The duckling is khaki-colored with pink beak and yellow feet. Adults are sepia with metallic green sheen (Figure 17.1). According to Hoffmann (1988, personal communication) there is a lot of variation in adult color, feet are yellow or white, bill is pink, and the eyes are pearl or yellow. He has seen them in Italy, Taiwan, and Brazil, but not in France or North America. Sepia is autosomal recessive. In the presence of N/N the phenotype is called tortora (turtle dove) and with N/n^+ it is smoky grey (Taibel, 1961).

Self-white (*P*). Three genetic kinds of white markings are known in muscovy ducks: self-white, Duclair piebald, and white head (canizie).

Taibel (1954c) was the first to explain the genetic basis for self-color vs. self-white. Using the gene symbol *P* (pigment), p/p was said to be white, P/p was black and white speckled, and P/P was black. To conform with present usage of gene symbols, designations of homozygotes should be reversed, so that the wild-type is p^+ and the white mutation is *P*.

The white spotting or speckling in heterozygotes is variable and it presents no uniformity of pattern. The self-whites frequently have a black spot on the head, but presence or absence in ducklings is not indicative of presence or absence in mature plumage.

The *P* gene is not homologous with recessive white in domestic ducks since hybrids from white parents usually are colored (Hoffmann, 1988, personal communication). There seems to be no equivalent in muscovy ducks of the dominant and recessive whites known in other poultry species.

Duclair piebald (*d*). Another kind of white markings is the one defined by Taibel (1956) as Duclair piebald, after a breed of domestic duck with an analogous pattern. It is autosomal recessive and has been assigned the gene symbol *d*. It results in a white bird with black on the head, back, saddle, and tail (Figure 17.1). Extent of the black markings is variable. Heterozygotes may have small white bibs according to Hollander and Walther (1962), raising a question of whether the mutation is truly recessive.

White head, canizie (*C*). The third variant of white markings is caused by an autosomal dominant gene (Taibel, 1958) symbolized by *C* (canizie or white head). In colored birds with the gene, the head and upper third of the neck are white, the bill is pink, and the eye is dark (Figure 17.1). This plumage pattern is characteristic of the closely related wild Asiatic species *Cairina scutulata*. According to Taibel (1958) and Hoffmann (1988, personal communication), the white head or canizie trait does not appear until after the juvenile molt and its expression in duckling down has not been identified.

White down. Snow-white down has been seen by Hoffmann (1988, personal communication) among normal yellow ducklings, but no genetic studies have been undertaken. White down has been described in domestic ducks (Krizenecky, 1961) and it has been found and studied genetically in chickens (Hutt, 1951). A similar mutation in muscovy ducks would not be surprising.

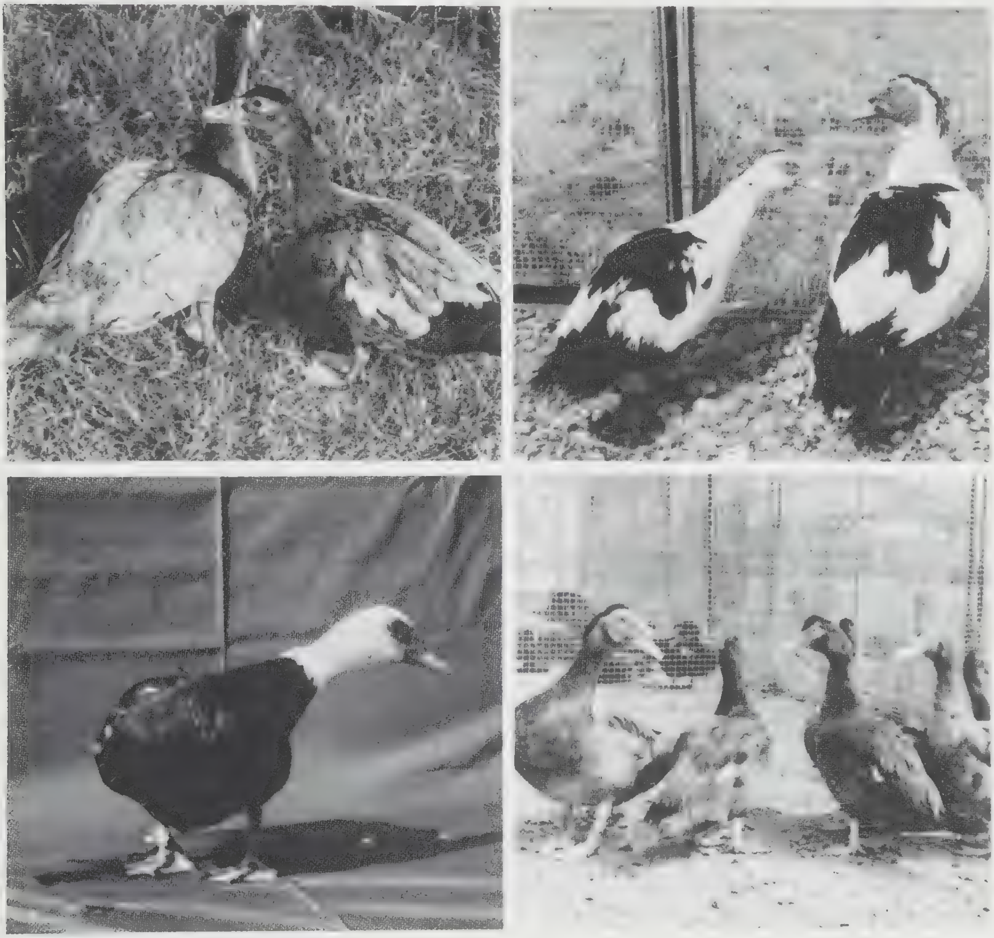

Figure 17.1. Four plumage color phenotypes in muscovy ducks. Upper left: blue dilution (N/n^+). Upper right: Duclair piebald (d/d). Lower left: white head or canizie (C/C). Lower right: sepia or faiogeno (f/f).

BLOOD AND BIOCHEMICAL VARIANTS

Limited research has been conducted on variations in blood constituents and in biochemical traits of muscovy ducks and hybrids with domestic ducks. Polymorphisms of antigenic factors on red blood cells and of serum prealbumins have been reported (McGibbon, 1944; Przytulski and Csuka, 1979). Refer to Chapter 8 for access to this specialized literature.

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Chapter 18. MUTATIONS AND MAJOR VARIANTS IN GEESE

Robert O. Hawes

The wild goose of central Europe is the western greylag (*Anser anser anser*), made famous by Konrad Lorenz. It is a brownish grey bird with buffy-white tipped feathers and with white under the tail. The eyes are brown surrounded by spectacles of orange skin; the bill is orange and the legs are flesh colored. A closely related subspecies, the eastern greylag (*A. a. rubrirostris*), has a pinkish bill with somewhat lighter feather color. There is general agreement among writers that the western subspecies is the ancestor of most present-day European breeds. This assumption is based on morphology, plumage pattern, and cytology (see Chapters 1 and 19).

The swan goose of central Asia (*Anser cygnoides*) is distinguished by a swan-like neck and a tendency for a knob at the base of the upper bill. A dark brown stripe starting at the head and extending down the dorsal side of the neck is a characteristic feature. Due to the similarity of plumage color between *A. cygnoides* and Brown Chinese and the prominent knob of the upper bill in both Brown and White Chinese, poultry writers have historically assumed that the swan goose is the ancestor of the Chinese breed. Cytological evidence supports this assumption (Bhatnagar, 1968).

The African breed has been an enigma for poultry historians. It probably originated in China, and not in Africa since no similar wild species is known there. It has been presumed to be a result of combining *A. anser* and *A. cygnoides*, since it exhibits color traits of the Chinese breed but has a body type similar to breeds derived from *A. anser*. Silversides et al. (1988) examined chromosomes of African and Pilgrim breeds and found a clear difference in the placement of the centromere of the fourth largest pair of autosomes. The African chromosome was metacentric, as it is in *A. cygnoides*, while that of the Pilgrim was submetacentric, as it is in *A. anser*. The F_1 offspring had one chromosome of each parental type. The African can now be classified as a descendant of *A cygnoides*, and more than a century of speculation laid to rest.

Darwin (1896) drew attention to the limited variation that is known in geese by writing that "hardly any other anciently domesticated bird or quadruped has varied so little". The Toulouse breed retains the plumage pattern and color of the western greylag; the African and Brown Chinese retain color and pattern of the swan goose. Color is changed in two western breeds, Pilgrim and Buff, but the greylag pattern remains. There are several white breeds. Only a few variants in other body systems are known. No lethal genes have been reported. Thus conservatism has been the way of the goose.

Genetic investigation of existing variants has been very limited. Part of the reason lies with long generation interval and with minor utilization of the species. Although thousand of goslings have been generated commercially from breed crosses, observations on their colors and morphology have mostly remained anecdotal. Since it now seems certain that domestic geese have descended from two wild sources, *A. anser* and *A. cygnoides*, geneticists can better proceed now to examine the variation that has accumulated. A first need is to determine inheritance of ancestral wild-type traits in crosses of the two species and of the breeds which have descended from them. Another need is to define more clearly the expression of variants in relation to the wild-type of both ancestral species.

COLOR AND PATTERN VARIANTS

The papers of Jerome (1953, 1954, 1959, 1970) present the first major genetic analyses of plumage colors and patterns. Earlier workers (reviewed by Jerome, 1959) described breeds and breed crosses, but they did not attempt genetic explanations. Jerome's work was based on crosses of White Chinese males with Emden and Toulouse females. He described six variants of color and pattern, assigning them to separate loci.

Autosomal recessive white (*c*). Birds of the genotype *c*/*c* are entirely white with orange bill and feet and blue eyes. Gosling down is yellow. It is characteristic of White Chinese. However the mutation seems not to be carried by western breeds except where it has been introduced from White Chinese. Hence in crosses of White Chinese with white-feathered western breeds, the progeny are usually found to be colored. And thus the mutant appears to be recessive to both *anser* and *cygnoides* wild-type (C^+).

Sex-linked recessive spotting (*sp*). Colored plumage is restricted to the head, upper neck, back, shoulders and thighs; eyes are blue. The term spotting is somewhat of a misnomer since the nonwhite areas retain the wild-type pattern. This pattern has traditionally been known as pied or saddleback. Jerome (1953, 1954, 1959) found it in the white-feathered Emden breed, and it is characteristic of the Grey Pomeranian. Its status in eastern breeds needs further study.

Sex-linked dominant dilution (*Sd*). White plumage in breeds descending from the greylag seems to be the result of a combination of the sex-linked genes for recessive spotting (*sp*) and dominant dilution (*Sd*), and not the result of the recessive white (*c*) gene. Thus the genotype for males is *Sd*/*Sd sp*/*sp* and for females *Sd*/- *sp*/-. The dilution gene has the ability to lighten the spotted pattern to white. Goslings of these breeds can be sexed with some accuracy, males having yellowish heads and grey backs, and females having greyish heads and grey backs. Juvenile and adult feathers show little melanic pigment although breed standards do allow for traces of grey in the back, wings, and tail of young specimens.

The combination of solid pattern (Sp^+) plus dilution (*Sd*) has produced the autosexing breed known as Pilgrim, in which males are white and females are grey. The genotype of the male is Sp^+/Sp^+ *Sd*/*Sd* and of the female $Sp^+/-$ *Sd*/-. It is assumed that two dilution alleles in the male are capable of preventing the formation of melanin pigment while in the female one dilution gene can only lighten, not whiten, the solid grey pattern. Goslings are easily sexed, males having yellowish down and females being olive-grey. At maturity males will still show occasional grey areas on the back, wings, and tail. Females show white areas around the bill, the upper neck, and occasionally on the breast. The eye color for males is greyish blue and for females it is dark grey or brown. The spotted pattern without dilution ($sp/sp\ sd^+/sd^+$) is a breed requirement of the Grey Pomeranian.

Use of the sex-linked dilution *Sd* for autosexing was outlined by Bondarenko et al. (1986) for crosses between grey and white breeds as well as between two white breeds. This is a unique way to utilize sex-linked genes and concurrently to acquire heterosis in the progeny. They found *Sd* to be characteristically present in Emden, Italian, Rhenish, and Slovakian White breeds. Stasko (1970) has also used the *Sd* gene for autosexing of crossbred geese. He used a white synthetic stock (GL-Iv-f line derived from Italian and Rhein breeds) as the sire, and the colored Landes breed as the dam.

Although a sex-linked color dilution locus (*Sd*) seems to have been adequately identified, further critical examination and research is needed since Jerome's (1953, 1954, 1959) matings were made in only one direction and no F_2 generation was examined.

Autosomal neck stripe (*Ns*). This trait is characterized as a dark brown stripe starting on the crown of the head and extending down the neck. It is a feature of the wild-type pattern in the swan goose and in the domestic Brown Chinese and African. Jerome (1953) reported that the trait was dominant and assigned the gene symbol *Ns*. However, no F_2 matings were made, and his interpretation needs further proof. The manner in which the F_1 matings were made indicates that the trait is autosomal in nature. When Vecchi (1930) crossed *A. cygnoides* x *A. anser* (whether wild or domestic was not specified), she found that all birds in the F_1 and F_2 generations exhibited neck stripes of varying intensity leading her to conclude that multiple factors were involved.

Recessive buff (*g*). This trait was reported to be allelic to wild-type grey (G^+) of western breeds (Jerome, 1954). It is expressed as a fawn shade of buff. There is no change in the feather pattern. The eyes are brown. It is sex-linked (Jerome, 1970). The color is a breed requirement of American Buff, Brecon Buff, Buff Pomeranian, and Buff Toulouse. Its action in *cygnoides* breeds is not known.

The author observed a buff mutant in a flock of tame Canada geese (*Branta canadensis*) in Prince Edward Island, Canada in 1987 and was informed by the owner that such color variants have been known in other flocks in the province. There was no change in the basic pattern.

Breast patch (*Wb*). This trait is expressed as a crescent-shaped white patch on the breast. It is not found as a standard characteristic in any present-day breeds. Jerome (1953) classified the trait as a dominant on the basis of an F_1 generation from a White Chinese male mated with a Toulouse female, and suggested the gene symbol *Wb*. Crawford (1988, personal communication) found a breast patch in the F_1 offspring of an African male x Pilgrim females and it was present in some F_2 birds. The author also found it in F_1 progeny from reciprocal crosses of Pilgrim and White Chinese, but not in progeny from Pilgrim and Roman. All of these observations suggest that breast patch originates with *cygnoides* breeds only.

The work of Jerome (1953, 1954, 1959, 1970) has provided an excellent start in describing genetics of plumage color in geese. But much more research and observation is needed before plumage colors and patterns can be adequately described in genetic terms.

Color of bill and feet. The western greylag has an orange bill with pink feet and legs, while the eastern greylag has a pink bill with pink feet and legs. Domestic geese descending from the greylag fall into two basic categories: they have either orange bills and feet, or pink (flesh-colored shading to pink or pinkish red) bills and feet. Birds with flesh-colored extremities tend to have white skin and flesh, those with orange extremities have yellowish skin. No definitive work has been done to determine the genetic relationship of these two phenotypes. Grow (1972) suggested that pink extremities are common faults in certain breeds requiring orange extremities (Emden, Pilgrim and American Buff) but also suggested that birds with orange bill and feet should be removed from breeds requiring pink extremities (Roman and Pomeranian). The British breed standard skirts the issue by allowing both pink and orange bill and feet in the Brecon Buff. It would appear that only the two color variations exist and presumably few genes are involved.

Since neither of the greylag subspecies possesses orange feet and legs as found in some domesticated varieties, it is interesting to note that the white-fronted goose (*A. albifrons*) may have been kept in the Old Kingdom of Egypt (2686-1991 B.C.) (Zeuner, 1963) and does possess orange feet and legs. Could it be that a bit of genetic information was exchanged between *albifrons* and *anser* which became incorporated into domestic stock? One other minor similarity between *A. albifrons* and domestic breeds is the white ring of feathers around the upper bill (hence, white-fronted) which occurs in the wild species and both in the Toulouse and in females of the Pilgrim breed, being a fault in the former and a breed requirement in the latter.

Delacour (1954) stated that the eastern greylag was probably never domesticated yet the pink bill found in that subspecies and in domestic forms causes speculation. Further cytological and biochemical genetic studies would help to answer these questions.

Bill colors in day-old goslings were observed by Jerome (1953) who related certain bill color types to specific down patterns. He reported that the dilution gene *Sd* had a profound effect in reducing melanin deposition.

Stasko (1970) reported a sex-linked gene pair which affected color of gosling bill and feet. The recessive gene (*b*) lightened the extremity colors. It apparently is different than sex-linked dilution (*Sd*) which was also present in his birds.

In a cross between an African male and Pilgrim females, Crawford (1988, personal communication) reported that bean color in male offspring was pink and in female offspring it was black. In F_2 and F_3 generations the traits appeared to be not sex-related.

PLUMAGE VARIANTS

There is a very obvious plumage difference between *A. anser* and *A. cygnoides*. The neck feathering of *A. anser* and of all its domestic descendants is crenellated or rippled while the neck plumage of *A. cygnoides* and its domestic descendants is smooth. No definitive work has addressed this difference, but casual observations by the author from various crosses between Chinese and other domestic breeds would suggest that the trait is polygenic.

Tufted. Two plumage variants have arisen in *A. anser* descendants. The Roman breed is presently recognized in two different forms, one is smooth-headed and the other has a small tuft of feathers on the head. The American Poultry Association breed standard recognizes only the tufted variety while the Poultry Club of Great Britain recognizes only the smooth-headed form. Grow (1972) stated that tufted geese have been known in many breeds and should not be designated only as a variety of the Roman. Information on the inheritance of the trait is unavailable; intuition would suggest incomplete dominance.

Sebastopol. The second trait is expressed as an elongation and spiralling of the plumage over most of the body excepting the head and neck, as seen in the Sebastopol breed. Even primary and secondary wing feathers are twisted and curled. Anecdotal information suggests that the trait is incompletely dominant and responds to mass selection for the intensity of curl demanded for exhibition purposes. Darwin (1896) recorded that the trait is transmitted to half-bred birds. The plumage is somewhat fragile and under crowded conditions becomes broken and ragged. Propagators of this breed, as well as writers of texts on waterfowl, attest to its extreme docility. It would be of interest to determine if this temperament is a pleiotropic effect of the plumage variation. This feather condition and frizzling in chickens are somewhat analogous.

ANATOMICAL VARIANTS

Three anatomical variants are known in geese. All are heritable but their genetic basis is not fully understood.

Knob. The small protuberance at the base of the upper bill found in the swan goose has been greatly exaggerated by artificial selection in the African and Chinese breeds. The wild greylag goose and its descendants lack this trait. By producing crosses between the two species and observing F_1, F_2 and backcross generations, Vecchi (1930) concluded that inheritance was dominant but due to numerous factors. Jerome (1953) suggested the symbol *Kb* and classified knob as an autosomal partial dominant without having the information that could be provided by an F_2 generation.

Angel wing. A second trait termed angel wing by waterfowl breeders has been known in domestic and muscovy ducks, swans, and geese for many years. The abnormality may be observed as a unilateral or bilateral twisting of the wing feathers. The bones of the hand and fingers are twisted laterally in their full length so that the distal end is turned out 45 degrees from the normal position. Suggested causes are heredity, weight of the growing feathers, rapid growth of feathers, and pecking by pen mates (Francis et al., 1967). Matings between normal birds yielded 14.7 percent abnormal offspring while matings between abnormal mates yielded 53.0 percent abnormal offspring. Thus, the conclusion was made that inheritance is not simple, but these results do suggest a genetic predisposition.

Dewlap. An anatomical requirement of the Toulouse and African breeds is the dewlap, a pendulous fold of skin attached to the rear of the lower bill and the front of the upper neck. The presence of a dewlap in the African breed has been used as evidence for its descent from greylags via the Toulouse. However, in early photos Brown Chinese are sometimes seen with small dewlaps which are of equal size to those of Africans of the same era (Brown, 1906). Thus the dewlap of the African may actually antedate that of the Toulouse. The trait in Toulouse is seen only in exhibition specimens, not in commercial stock. It would appear to be quantitative in its inheritance.

BIOCHEMICAL VARIANTS

Egg lysozymes. A type of lysozyme, designated *c* has long been known to occur in the egg white of chickens and many other galliform species as well as in the egg white of some anseriform species (Prager and Wilson, 1974). In 1967 Canfield and McMurry reported a new type of lysozyme, designated *g*, in the Emden goose. Prager and Wilson (1974) have since detected the *g* type of lysozyme in egg whites of species representing nine different orders of birds. They discuss the implications of the presence of the *c* and *g* types but point out that the presence or absence of these lysozymes in egg white does not necessarily reflect the presence or absence of the genes which code for these proteins. Arnheim and Hindenburg (1973) reported that both the *c* and *g* types are found in the black swan (*Cygnus* *atratus*). They suggested that the goose-type and chick-type lysozymes are the products of two distinct loci and discussed possible genetic mechanisms for the distribution of the two types of lysozymes.

Transferrins and conalbumins. Early work by Markus and by Valenta and Stratil described polymorphic proteins in goose serum, egg white, and egg yolk (see Valenta and Stratil, 1978). Baker and Hanson (1966) examined the transferrin patterns in eleven species of geese (eight of *Anser* and three of *Branta*). The patterns showed a close relationship between the two genera.

In 1978, Valenta and Stratil analyzed blood serum, egg white and egg yolk from four domestic descendants of *A. anser*, the White Bohemian, Roman, Landes, and Rhineland breeds. Three transferrin and conalbumin phenotypes were detected. They concluded that the transferrins in their study were determined by one locus having two codominant alleles Tf^A and Tf^B. They also concluded, in agreement with others, that the transferrins and conalbumins are genetically controlled by the same locus. The presence of the two alleles Tf^A and Tf^B was reported by Przytulski and Blakala (1981) after examining serum samples from the Pomeranian breed.

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Chapter 19. CYTOLOGY AND CYTOGENETICS

J. James Bitgood and Robert N. Shoffner

Cytogenetics is the study of chromosomes. It emerged as a separate field of poultry genetics in the early 1960s, and has provided another tool for studying inheritance in birds. The chicken is an acceptable model for cytogenetic research since its chromosomal behavior parallels that of other species. Principles of meiosis and mitosis apply to birds as well as to other species.

Embryos for chromosomal analyses can be easily recovered without surgical techniques. Large numbers of embryos and hatched progeny can be obtained from one planned mating. Feather pulp is a rapid and repeatable source of metaphase plates for chromosome analysis without contamination problems associated with cell cultures. Cell culture, cell hybridization, banding methods, radioactive labelling, and other techniques are readily applicable.

Considerable differences existed in early reports about the number, morphology, and function of chicken chromosomes. Hutt (1949) summarized findings and Brant (1952) reviewed the difficulties in working with avian chromosomes. Birds have two loosely defined subsets of chromosomes. Macrochromosomes are the larger identifiable chromosomes, and microchromosomes are the greater number of small ones, nearly all individually unidentifiable (Figure 19.1). This morphology is ubiquitous among the over 200 avian species so far karyotyped, and also among reptiles, the ancestors of birds.

This chapter reviews information on avian cytology and cytogenetics. Special emphasis is on the chicken because most studies have been conducted on this species. Findings in other avian species will be cited where available. Evidence supports the concept of conservation of genetic processes within avian species. Principles learned from one species will most likely apply to other species.

Chromosome numbers of the eight species discussed in this book are given in Table 19.1. Because of microchromosome identification difficulties, reported numbers in a species may change in later reports. Refinements to the numbers in this table should be expected as techniques are improved.

THE MICROCHROMOSOMES

At one time a diversity of opinion existed as to whether the microchromosomes were regular chromosomes. Newcomer (1957) labelled them as 'chromosoids' to distinguish them from what he felt were the true chromosomes. Ohno (1961) referred to them as microchromosomes. Schmid (1962) and Donnelly and Newcomer (1963) reported that the uptake of ^{3}H was similar in both the microchromosomes and the macrochromosomes. Ford and Woollam (1964) used both light and electron microscopy and could see no size gaps, no noticeable structural differences, and no behavioral differences during either meiosis or mitosis. Bloom and Buss (1967, 1969) reported that at the level of the light microscope, both types of chromosomes reacted similarly to ammoniacal silver and Feulgen stains. Clement (1971) reported that the microchromosomes showed the same radioactive labelling as other chromosomes.

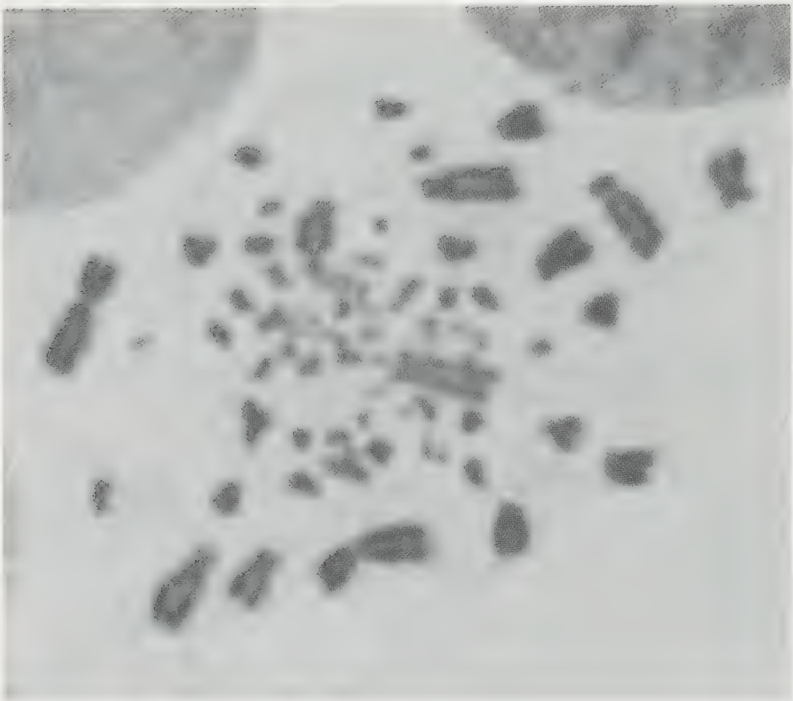


Figure 19.1. Mitotic chromosome spread from a turkey. The large number of small dark staining objects are the microchromosomes that diminish in size to the smallest that are barely discernible under the light microscope. (Photograph courtesy J.S. Otis).

Table 19.1. Chromosome numbers of the species reviewed in this book.

Species	2*n*	Reference
Chicken	78	Beçak et al. (1971)
Domestic duck	80	Beçak et al. (1973)
Muscovy duck	80	Migliore et al. (1986)
Goose	80 *	Hammar (1966)
Guinea fowl	78	Beçak et al. (1971)
Ring-necked pheasant	82	Beçak et al. (1971)
Japanese quail	78	Beçak et al. (1971)
Turkey	80	Beçak et al. (1971)

* Hammar (1966) reported 80 chromosomes in the European greylag goose, the supposed ancestor of western breeds of geese, and 80 in the Asiatic swan goose, the supposed ancestor of the Chinese and African breeds. However, Beçak et al. (1973) reported the Asiatic swan goose had 82+ chromosomes. Beçak et al. (1975) showed some species of geese with 80+ chromosomes, and others with 82+. The numbers need verification.

There are functional genes on the microchromosomes. Kao (1973) fused Chinese hamster and chick cells and found that the locus for *Ade-A* was on chromosome 6 of the chicken, and the locus for *Ade-B* was on chromosome 7. Leung et al. (1975) located the locus for thymidine kinase on a microchromosome smaller than the ones Kao had mapped. The sequences related to the transforming gene of avian sarcoma virus are located on an unidentified microchromosome (Padgett et al., 1977).

Ohno et al. (1962) postulated that some of the microchromosomes in birds functioned as nucleolar organizers. It is now known that the NOR is on a single chromosome in the chicken. Hughes et al. (1979) used hybridization techniques and found that most of the ribosomal cistrons were located on chromosomes 10-15. Bloom et al. (1978) and Bloom and Bacon (1985) investigated chickens trisomic for a microchromosome and located the major histocompatibility complex (MHC), which is the B blood group or erythrocyte alloantigen B (*Ea-B*), and the nucleolar organizing region (NOR) on a microchromosome in the 15-18 size range. Auer et al. (1987) reported that the NOR is located on chromosome 17 in the chicken.

The NOR is the chromosome region that contains the genes that transcribe ribosomal RNA. There are about 145 ribosomal gene repeats in each NOR (Muscarella et al., 1985). These genes are highly repetitive and the collective activity of these genes forms the nucleolus. It is composed of RNA, associated proteins, and at least some of the enzymes needed for assembly of the ribosomes. In a normal diploid chicken, a maximum of two nucleoli per nucleus are visible. In triploid chickens there are three NORs per nucleus (Bloom et al., 1969). Three NORs are also a distinguishing feature of chickens trisomic for chromosome 17 (Bloom and Bacon, 1985; Macera and Bloom, 1981).

In a study of synaptonemal complexes formed during meiosis in chickens, Kaelbling and Fechheimer (1983a) identified centromeres on all chromosomes, including microchromosomes. They later (1983b) showed microchromosome involvement in several translocations. Blazak and Fechheimer (1980) showed a microchromosome involvement in a t(Z;micro) reciprocal translocation. Wang et al. (1982) and Wooster et al. (1977) suggested that microchromosomes were involved in other translocations. Hutchison (1987) demonstrated that microchromosomes form lampbrush chromosomes that are similar to macrochromosomes.

All evidence indicates that avian microchromosomes are true chromosomes, differing from other chromosomes only in size and possibly in the proportion of repetitive DNA in the chromosome. Arbitrary separation of macro- and microchromosomes has been made by various investigators, the dividing point ranging from chromosome pair 5 to 9 or 10. Occasionally even a smaller size has been used, depending on the focus of the particular researcher. With no generally agreed upon standard to distinguish them, the separation of macro- and microchromosomes will continue to be arbitrary.

AVIAN SEX CHROMOSOMES

The avian female is the heterogametic sex, ZW, and the male is the homogametic sex, ZZ, as contrasted to mammals where the male is the heterogametic sex, XY, and the female is the homogametic sex, XX.

The Z-W nomenclature apparently was originally used to indicate that this was a different sex chromosome system than the X-Y system found in mammals and *Drosophila*. It was somewhat prophetic, in that current evidence indicates that there is very little homology in genetic coding content as well.

The ZW and XY sex chromosome systems may have evolved separately. This agrees with the statement by Morizot et al. (1987) that evidence "... suggests that evolution of sex chromosomes may have been accomplished independently on multiple occasions in the vertebrates." Beçak et al. (1964) proposed that snakes and birds originated from the same lineage and the ZW sex chromosome evolved as a part of that lineage. Mammals appeared later, and the XY chromosome system evolved as a part of that evolutionary chain.

Some genes that are sex-linked in mammals are autosomal in birds and vice versa. Bhatnagar (1969) found that the locus for glucose 6-phosphate dehydrogenase (G6PD) is autosomal in chickens and ring-necked pheasants, whereas it is X-linked in mammals. The enzyme aconitase is autosomally inherited in mammals, but Z-linked in birds (Baverstock et al., 1982). Cam and Cooper (1978) found that phosphoglycerate kinase (PGK) exists in only one autosomal form in the chicken. Mammals have one sex-linked form (PGK-A) and a second autosomal form (PGK-B) that is found only in sperm. No PGK is found in chicken sperm. Cam and Cooper (1978) reviewed the literature on G6PD and ornithine transcarbamylase, both of which are X-linked in man, but autosomal in birds. They concluded that there is no evidence of homology between the mammalian X chromosome and the avian Z chromosome. However, relatively few loci have been screened for possible homology.

Spillman (1908) first suggested the heterogametic nature of the female chicken. Later reports stated that the sex chromosome was one of the largest or the largest in the chicken. However, Hutt (1949) reviewed the literature that identified the fifth largest chromosome as the chicken sex chromosome. Painter and Cole (1943) stated that one chromosome, the fourth (their *d*) or the fifth (their *e*) "...is unpaired in the female pigeon." They also stated that "...five other markers...show...in the female...that it is either the *d* or *e* chromosome which is without a mate."

Werner (1927) seems to be the first to use the Z-Z symbolism to designate the male avian sex chromosomes, and the Z-W symbolism for the female sex chromosomes. Suzuki (1930) is cited by Hutt (1949) as being the first to correctly identify the fifth largest as the sex chromosome. Sokolow et al. (1936) also identified the fifth largest as the sex chromosome and mentioned that the female was heterozygous, with sex determination following the ZO or ZW pattern. Miller (1939) also applied the Z nomenclature to the fifth largest chicken chromosome.

In most avian species, the Z chromosome is either the fourth or the fifth largest chromosome. Often when the Z is the fifth, it is metacentric. In many cases when the Z is the fourth, it is subacrocentric (Beçak et al., 1971). According to Ohno et al. (1964) the Z chromosomes from six avian species and one hybrid were all similar in size. G-banding suggested that this was due to a pericentric inversion. It can be hypothesized that chromosomal inversions have occurred during evolutionary time to create the morphological differences seen in Z chromosomes.

Some early investigators thought that the female had only a single sex chromosome and labelled this situation as ZO. The settlement of the ZO versus the ZW question came with a burst of papers, each contributing to the understanding of the nature of the Z and the W chromosomes in birds. The critical paper on the avian W chromosome was by Frederic (1961). Schmid (1962) stated that Frederic identified the W chromosome as an unpaired chromosome among the larger microchromosomes in female cells. Schmid utilized autoradiography on cultured chicken cells, and found that the Z chromosomes in male chickens, the fifth largest pair, replicated DNA in the same manner as the other macrochromosomes. The single Z chromosome in the female chicken also followed this same pattern, but one of the larger microchromosomes underwent late replication, comparable to the mammalian Y chromosome. Schmid indicated that this microchromosome was identical to Frederic's W chromosome.

Rothfels et al. (1963) identified the W chromosome in the budgerigar. It was approximately metacentric in configuration and in the size range 9-10. Other papers demonstrated the presence of a W chromosome in avian species: Ohno et al. (1964), canary and pigeon; Owen (1965), chicken; Krishan et al. (1965), great horned owl; Krishan and Shoffner (1966), chicken, turkey and Chinese pheasant; Galton and Bredbury (1966), pigeon; Stephos and Arrighi (1971), chicken, parakeet, ringed turtle dove, pheasant, duck, bobwhite quail and chukar partridge; Au et al. (1975), bald eagle; and Biederman et al. (1980), great horned owl. The C-band technique positively identifies the W chromosome and in all species where it has been applied the W chromosome has been documented.

Analysis of the W chromosome at the molecular level is revealing information on its structure. The W chromosome is heterochromatic, with a highly repetitive DNA content. There is evidence for a W-linked histocompatibility locus (see Chapter 21, and Bloom, 1974). Tone et al. (1984) identified two repeating DNA units in the chicken W chromosome. A 0.6 kb unit was repeated approximately 14,000 times and a 1.1 kb unit approximately 6,000 times, both repeat units accounting for about 46 percent of the W DNA. These same repeated units were found in three junglefowl species (*G. gallus, G. sonnerati, and G. varius*) but not in the Japanese common pheasant, guinea fowl, Japanese quail, or the rock dove. It was suggested that the 0.6 and 1.1 units were present in a common *Gallus* ancestor. Msp I sites associated with the 0.6 kb fragment in *G. varius* were different from the other two junglefowl species, suggesting that *G. varius* is not as closely related to the other junglefowl as they are to each other.

Morizot et al. (1987) reported that the locus for muscle creatine kinase in Harris' hawks was sex-linked, with evidence for both Z- and W-linked alleles. Results from similar work in other birds would be of interest.

Bloom (1970b) reported an embryo with a 3A-ZZZW chromosome complement, indicating that the sex chromosomes also undergo nondisjunction. Mayr and Auer (1988) found a 2A-ZWW viable turkey, however no other studies were conducted because the bird was slaughtered before the tissue sample was analyzed. A comprehensive review of literature on the avian W chromosome was made by Bloom (1974). He concluded that the majority of bird species have a ZZ-ZW sex determining mechanism and that the W may have some genetic activity. Subsequent reports have substantiated these conclusions.

Sex determination. Abdel-Hameed and Shoffner (1971) showed that intersex chickens often are ZZW triploids. Occasionally one will be mosaic for ZZ and ZW diploid cells. Thorne et al. (1987) found that the fertility of viable mosaic chickens depended on sex chromosome complements and ratio of diploid to triploid cells. Diploid-triploid chickens with a low level of triploid cells had the diploid phenotype, and were fertile regardless of the sex chromosome complement of the triploid cells. Intersexes were found when a higher proportion of triploid cells were present.

The W chromosome has a major influence on determining femaleness. No ZW diploids with male or intersex phenotypes have been reported. ZZW triploids and individuals with other sex chromosome mosaicism have different degrees of female phenotypes. No ZZ or ZZZ males have been found with female phenotypes.

Dosage compensation. It appears that dosage compensation as seen in mammals does not occur in birds. Evidence indicates that neither of the avian Z chromosomes undergoes inactivation in the homogametic sex (the male). This is of interest in regard to the evolution of heteromorphic sex chromosomes. Separate ZW and XY sex chromosomes might have arisen first, followed by the mammalian X-inactivation mechanism.

Several mechanisms have evolved to adjust the level of transcripts produced by genes on the sex chromosomes of some species. Mammals use X chromosome inactivation. In *Drosophila*, the genes on the single male X chromosome increase the quantity of product relative to each of the female X chromosomes. There is an apparent reduction of activity of the same gene in the female fly on each X chromosome (reviewed by Ohno, 1966). Evidence suggests that birds are not concerned by a difference in the amount of Z chromosome gene product produced by each sex. Among the gene loci studied to date, the males apparently produce more product than the females because of the two active Z chromosomes.

The classical example in the chicken is the Z-linked barring gene. This gene creates a horizontal white (nonpigmented) bar on what would otherwise be a pigmented feather. The double dose of this gene in the ZZ male results in greater pigment restriction than in the ZW female. The heterozygous male exhibits a barring pattern similar to that of the hemizygous female, with a narrower white bar than the homozygous male with its double dose of the gene. Cock (1964) lists a number of examples in poultry where Z-linked dosage compensation does not appear to exist.

Further evidence for the lack of dosage compensation is being provided by molecular and biochemical studies. Lasher and Bitgood (1982) found that the Z-linked recessive white skin mutation (y) (McGibbon, 1981) affected blood plasma coloration. Homozygous males and hemizygous females show clear plasma, rather than the normal straw-colored plasma. The color of plasma from heterozygous males is somewhat intermediate between homozygous and normal males. Baverstock et al. (1982) found that the enzyme liver cytosolic aconitase is Z-linked in the guinea fowl, and probably in the domestic fowl, house sparrow and cockatoos, and does not exhibit compensation.

Goldman et al. (1987) suggested that specific signal or recognition sequences on the mammalian X chromosome are required for inactivation. These sequences would be spaced at intervals along the length of the chromosome. In the absence of a sequence, the region that the sequence would normally control would escape inactivation. They inserted the chicken transferrin gene into the X chromosome of a mouse, and found that the gene was not inactivated when it was on the inactive X chromosome. The avian Z chromosome apparently lacks these inactivation sequences. Also, the gene may have been inserted at such a location that it was beyond the range of control of the nearest inactivation sequence.

Bloom and Bacon (1985) reported that birds trisomic for the MHC will express all three B-haplotypes when three different alleles are present.

Muscarella et al. (1985) reported that rRNA levels in chicken fibroblasts from embryos disomic, trisomic, and tetrasomic for the NOR were similar even though gene copy number differed. This indicates that a type of dosage compensation occurs to maintain rRNA at the diploid level.

SEX RATIOS

The primary sex ratio (at fertilization) in the chicken is apparently equal between males and females. In studies of chromosomally normal matings, Fechheimer et al. (1968) examined embryos at 16 h incubation, Keller (1972) examined embryos at 60-90 h, and Fechheimer and Jaap (1973) examined embryos at 16-18 h. The evidence from these experiments was consistent with the hypothesis of a 1:1 primary sex ratio in early embryos. Dinkel et al. (1979) and Bitgood (1980) examined sex ratios in 24 h embryos from parents carrying chromosomal rearrangements, and found no significant deviation from a 1:1 sex ratio. In these studies the rearrangements had no effect on the sex ratios.

Byerly and Jull (1935) reviewed studies that investigated secondary sex ratios (at the time of hatching) among 18,000 embryos. An apparent excess of females died between the ninth day of incubation and hatching time. This same trend was noted by Fechheimer et al. (1968). In 266 embryos examined at nine days of incubation 49.2 percent were male, whereas in 269 embryos at 19 days of incubation 55.0 percent were male. Because of the relatively low numbers, there was not a significant difference from a 1:1 ratio, but the finding is consistent with the results of Byerly and Jull (1935).

There may be differential mortality during embryonic development, which means more males should hatch. There are no current studies on this. Hutt (1949) cited the summary by Landauer and Landauer (1931) of 48.77 percent males among 67,993 chicks. Hutt suggested that there may also be breed or strain differences.

MEIOTIC CHROMOSOMES

The meiotic process in both sexes of birds parallels that of other eukaryotic species, and will not be reviewed in this chapter.

Because of the large number of microchromosomes, the most accurate method for chromosome counts is to examine the male meiotic chromosome complement (Figure 19.2). The formation of the tetrad during early meiosis halves the number of structures and each chromosome is thicker than its mitotic counterpart, permitting easier examination

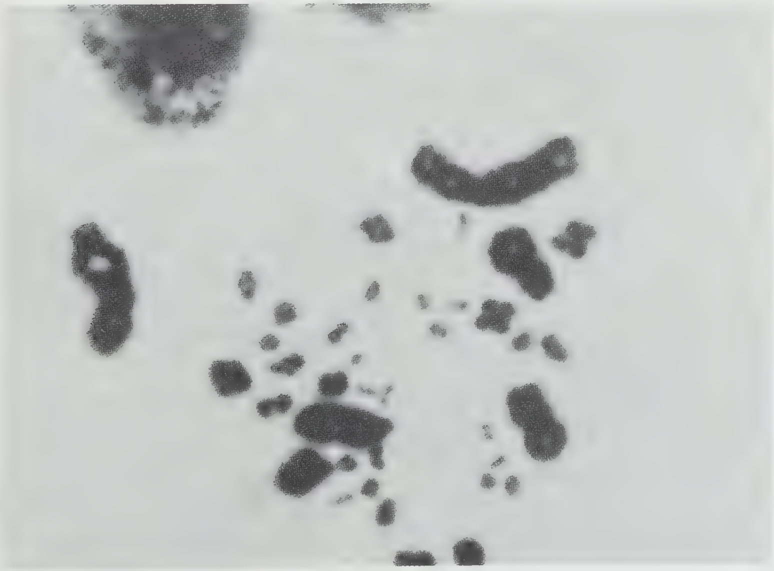


Figure 19.2. Diakinesis of a male chicken. Note the high number of chiasmata on the largest chromosome. Evidence suggests a high recombination rate in this chromosome. (Photograph by Andrea Jaccobuzzi courtesy J.S. Otis).

under a light microscope.

Ohno (1961) studied meiotic configurations in both sexes. The ZZ bivalent in the male behaved the same as autosomal bivalents of the same size. The single Z chromosome in the female did not exhibit positive heteropycnosis (dense, tightly-coiled) as in the XY bivalent of mammals. He found that oocytes in chicks are in diplotene in late embryonic or early postnatal life, as with mammals. Ford and Woollam (1964) examined chromosomes from testicular samples of mature males with light and electron microscopy, and found a continuous size series in meiotic chromosomes. The microchromosomes appeared and behaved the same as the macrochromosomes, including the presence of synaptonemal complexes.

After an intensive study of spermatogenic cells, Pollock and Fechheimer (1978) reported 39 bivalents and between 56 and 66 chiasmata in these bivalents. If each chiasma represents 50 percent recombination, the total genetic map of the chicken is at a minimum between 2800 and 3300 map units. A summation of the distances on the chromosome maps in Chapter 21 accounts for only about 515 map units.

There apparently have been no cases of segregation distortion noted in birds. This occurs when there is unequal genetic segregation between the two alleles present in a heterozygote.

Synaptonemal complexes. The synaptonemal complex (SC) was first described by Moses (1958), and von Wettstein et al. (1984) reviewed its function during meiosis. It is a proteinaceous-RNA structure that forms between homologous chromosomes during the pachytene stage of meiosis. This complex is found only in meiotic cells and is most easily visualized through an electron microscope. It is necessary for recombination, but its presence does not ensure that recombination will take place. The SC forms a framework that appears to align and stabilize the homologous chromosomes at a fixed distance from each other. It has been suggested that selected stretches of chromatin penetrate the SC and engage in molecular pairing. Coleman and Moses (1964) sectioned testes from male chickens and found that elements of the SC do not contain an appreciable amount of DNA. A thin thread of electron-dense material was seen between the paired pachytene chromosomes by Wylie (1972).

A microspreading technique used by Solari (1977) on female meiotic cells revealed 38 SCs and an unequal SC identified as the ZW pair, confirming earlier mitotic counts of chicken chromosomes. There is an homologous pairing segment on the Z and W chromosome (Solari, 1977; Rahn and Solari, 1986), as is the case with the mammalian X and Y chromosomes. Solari (1977) noted a lack of heteropycnosis in the ZW sex chromosome bivalent, as did Ohno (1961), which is in contrast to the heteropycnotic XY bivalent in mammals.

The segment of the chicken Z chromosome that pairs with the W chromosome (Solari, 1977) was found to be on the short arm of the Z chromosome (Solari et al., 1989). SCs from oocytes of day-old female chicks carrying one of two Z-linked chromosome translocations, the MN t(Z;1) and the OH t(10), were examined. The MN t(Z;1) break point (Wang and Shoffner, 1974) is on the long arm of the Z chromosome (Figure 19.3), and the OH t(10) break point is on the short arm of the Z chromosome (Telloni et al., 1976; Wooster et al., 1977) (Figure 19.4). The arm containing the OH t(10) break point was found to be the arm that paired with the W chromosome. This study provided genetic and cytological correlation of the arms of the nearly metacentric Z chromosome.

Kaelbling and Fechheimer (1983a, 1983b) studied SCs from spermatocytes of normal chicken males and heterozygous males from five single and one double chromosomal rearrangement mating. They recognized 39 chromosome pairs, and described the chromosome complement based on relative lengths of each chromosome and the centromeric index (CI). The CI is the length of the shorter of the two arms divided by the total length of the chromosome, multiplied by 100. They could not assign all microchromosomes to a specific size order because of similarities in lengths and centromere positions. They did map break points in the chromosome rearrangements to specific locations on the affected arms by measurements of the SCs.

Rahn and Solari (1986) examined recombination nodules (RN) in chicken oocytes. RNs are round or oblong structures associated with SCs correlated with crossing-over. All SCs, including the microchromosomes, contained at least one nodule. The ZW bivalent also contained a strictly localized nodule in the pairing end of this bivalent, suggesting that recombination may occur in this region (Figure 19.5). This obligatory RN suggests that there are 'pseudo-autosomal' Z- and W-linked genes. As these genes are identified, they can be used to study their effect in ZZ and ZW backgrounds. One candidate is the muscle creatine kinase locus. Morizot et al. (1987) reported Z- and W-linked alleles for this enzyme in Harris' hawks.

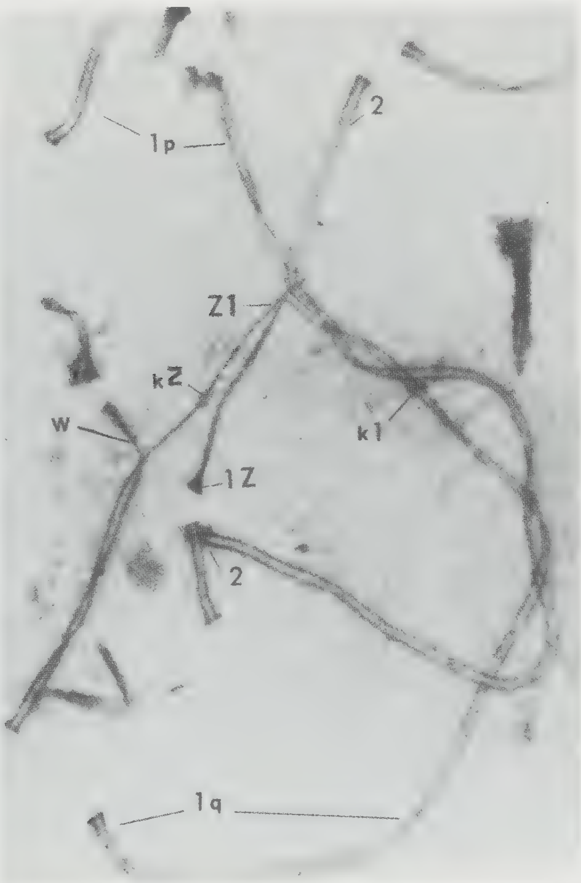
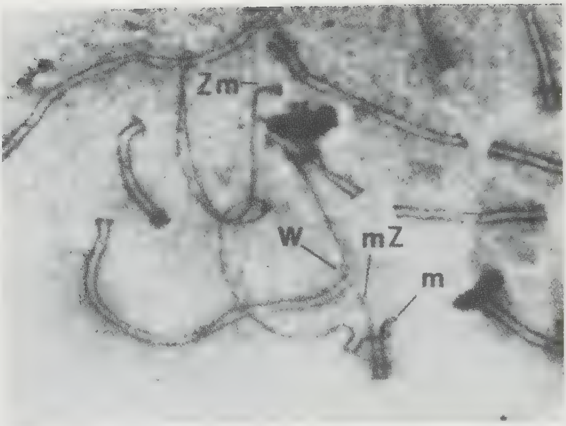


Figure 19.3. Synaptonemal complex formed by the MN t(Z;1) in a hemizygous female. The pairing between the short arm of the Z and the W is intact and not interrupted by the translocation break point. (Photograph courtesy A.J. Solari and N.S. Fechheimer).

Figure 19.4. Synaptonemal complex formed by the OH t(10) (m; microchromosome). The break point is on the short arm of the Z chromosome, and the disruption of pairing can be seen. (Photograph courtesy A.J. Solari and N.S. Fechheimer).



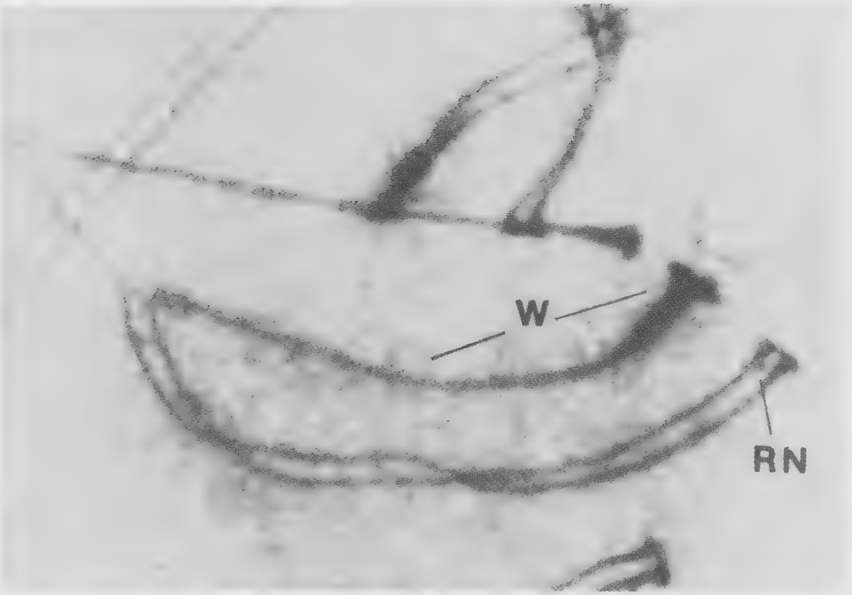


Figure 19.5. The recombinant nodule at the distal end of the Z-W bivalent in the chicken. This suggests that there may be genes homologous to both the Z and the W chromosome that would show pseudo-autosomal inheritance. (Photograph courtesy A.J. Solari and N.S. Fechheimer).

Lampbrush chromosomes. The term 'lampbrush chromosomes' (LBC) describes a stage of chromosome activity during diplotene of oogenesis (Figure 19.6). LBC contain regions of condensed chromatin, alternating with fibrous loops extending from the axis. These loops represent active transcription of RNA from the unravelled DNA template. The high level of activity suggests that these segments produce large amounts of mRNAs for use by the ovum and early zygote. The zygote would then need only to activate developmental genes, leaving housekeeping functions to RNAs derived from LBC. This transcriptional activity might also be preparation for sperm entry and fertilization, pronucleus formation, or other early developmental functions.

Ahmad (1970) reported LBC in chick ovaries at 18 days of age. He cited reports dating from 1904 that recognized LBC in the chicken. Wylie (1972) found LBC in birds from ten weeks posthatching to 22 weeks of age. Hutchison (1987) reported that chicken LBC are similar to LBC in newts and salamanders. She reviewed investigations on chicken LBC, and suggested that they may be suitable for microchromosomal gene mapping using pulsefield electrophoresis.

CHROMOSOME REARRANGEMENTS

Translocations. Translocations can be recognized by morphological differences from standard chromosomes unless equal lengths of chromosome arms are exchanged. The longitudinal or cytochemical differentiation banding procedures (Hsu, 1973) are commonly used to identify equal exchanges.

The first reported avian chromosome translocation was induced in a chicken by X-rays, with break points on chromosomes 1 and 2 (Newcomer, 1959). Several laboratories

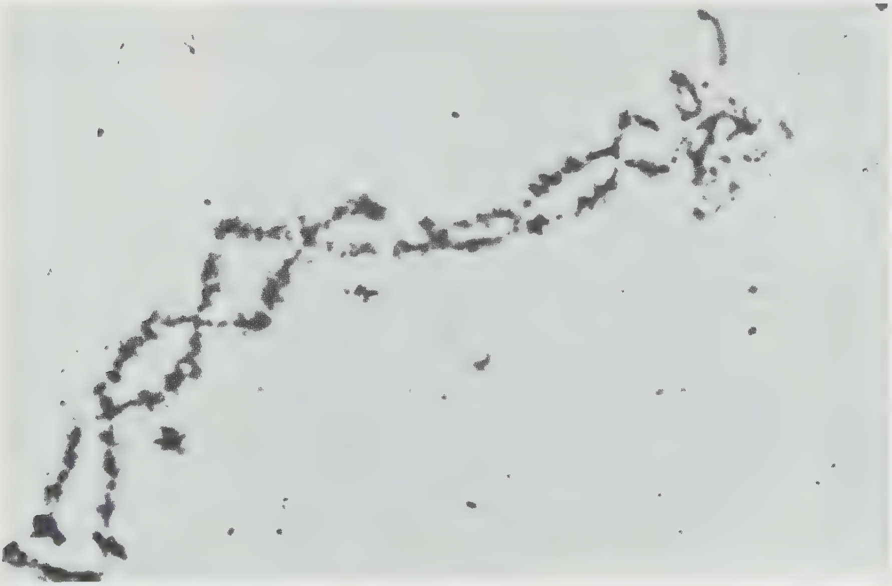


Figure 19.6. An example of a lampbrush chromosome from an avian oocyte. The filamentous structure is indicative of active RNA transcription. (Photograph courtesy N. Hutchison).

induced translocations in the early 1970s, using two methods: X-irradiation of semen (Zartman, 1971; Wooster et al., 1973), and injections of mutagenic chemicals into mature males (Shoffner, 1972). A number of translocations have been established in breeding lines of chickens (Wooster et al., 1977; Wang et al., 1982).

Two spontaneous translocations have been reported in chickens. One was an exchange between chromosomes 2 and 3 in a Single Comb White Leghorn (Ryan and Bernier, 1968). The other was an exchange between chromosome 2 and a microchromosome (Shoffner, 1982).

In contrast to plants where rearrangements in the heterozygote create semisterility, animal gametes with duplication and deficient (dp/df) chromosomes are capable of fertilization. Chromosomally unbalanced gametes from either sex of the mouse can participate in fertilization (Ford, 1971). Blazak and Fechheimer (1979) and Bitgood (1980) independently demonstrated that unbalanced gametes from chickens of either sex are capable of fertilization (Figure 19.7). About 20 chicken chromosome rearrangement lines have been developed. Heterozygous translocation carriers are fully fertile; repeatable instances of 100 percent fertility in a significant number of controlled matings have been found (Bitgood and Shoffner, unpublished). One exception (Shoffner, unpublished) is an induced translocation between the long arm of chromosome 2 and an unidentifiable microchromosome, wherein the homozygous male produces abnormal spermatozoa incapable of fertilization, but heterozygous males and both homozygous and heterozygous females have normal fertility.

Females hemizygous for Z-linked rearrangements produce a portion of gametes with dp/df chromosomes as do individuals heterozygous for autosomal translocations. These are

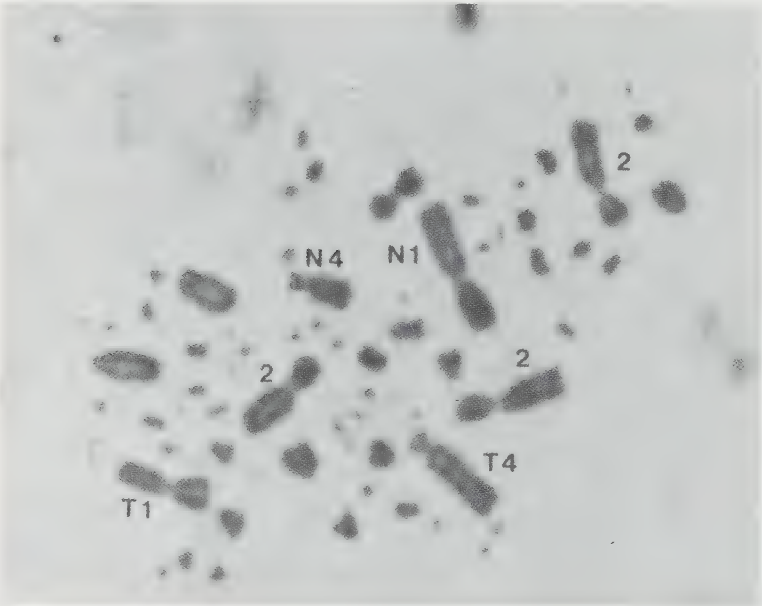


Figure 19.7. An embryo trisomic for chromosome 2. It resulted from nondisjunction in a carrier of the MN t(1;4) translocation. Trisomy for a chromosome not involved in the translocation may or may not be a fortuitous event. (Photograph courtesy J.S. Otis.).

produced by adjacent meiotic segregation (Burnham, 1956; reviewed by Blazak and Fechheimer, 1979). These gametes result in death to about one-half of the early zygotes.

Male chickens with either Z-linked or autosomal translocations are fertile and when homozygous reproduce with no abnormal zygote loss. In contrast, male mice carrying X-linked translocations are sterile (reviewed by Ashley, 1983). Chickens are a more favorable model system to study sex-linked translocation behavior because segregations in offspring can be documented.

There are no phenotypic changes associated with chicken translocations. The embryos with unbalanced chromosome complements die. In mammals, in addition to embryonic mortality, there are examples of viable offspring with an unbalanced chromosomal complement that exhibit phenotypic aberrations.

An unusual chromosomal translocation induced by Wooster et al. (1977) and studied by Bitgood et al. (1981) had breaks in three chromosomes, with the fragments annealing in a cyclical pattern. The breaks were induced in chromosomes 1, 5, and 8. The fragment from 1 joined the intact portion of 8, the fragment from 8 joined the intact portion of 5, and the fragment from 5 joined the intact portion of 1. This was the first cyclical translocation (as defined by Burnham, 1956) reported in any vertebrate.

Translocations are useful as markers for parental origin in primordial germ cell transfer, parental origin of gametes, and cell fusion studies. They have been used to map genes to specific arms of chromosomes and to map centromeres. Translocations have also been used to study meiotic segregation of chromosomes. As in other species, they have undoubtedly been of importance in evolution. Shoffner et al. (1979) suggested that the difference in chromosome morphology seen in the Ross's and emperor goose was due to a translocation between chromosomes 1 and 3, perhaps a factor in speciation.

Inversions. Thornycroft (1966) found inversion polymorphisms in populations of white-throated sparrows. Shields (1973, juncos) and Bass (1979, cardinals) found other inversion polymorphisms, suggesting chromosome evolution. The question remains as to which morphs are original and which are the nascent ones in these polymorphic populations.

Paracentric inversions are difficult to recognize, requiring banding procedures, examination of meiotic tissues, or breeding tests. A paracentric inversion involves only one arm of a chromosome, reversing the gene order without altering the length of the arm. None has been identified in domestic birds.

Two induced pericentric inversions have been studied in the chicken, each with unique features. The chromosome 1 inversion, inv(1), was induced with ethyl methanesulfonate (Wang and Shoffner, 1974). It has not been recovered in homozygous individuals, indicating either a position effect or loss of a DNA sequence. Approximately 50 percent early embryonic mortality occurs when heterozygous individuals are mated to normal individuals. This is expected due to creation of duplication and deficiency chromosomes from recombination between the normal and inverted chromosomes. Bitgood et al. (1982) observed that some progeny from heterozygous *inter se* matings carried two chromosomes morphologically distinct from either the normal or the inversion chromosome. These were recombinant chromosomes, rec(1), generated when crossing-over occurred in the inversion loop. The rec(1) carriers appear normal and are fully fertile as adults. When rec(1) individuals are mated, normal, inv(1) heterozygotes, and rec(1) heterozygotes are recovered. Insufficient matings have been conducted to determine the proportion of genotypes produced from the various mating types.

In a study of the inv(1), Bitgood (1980) found embryos homozygous for the inversion

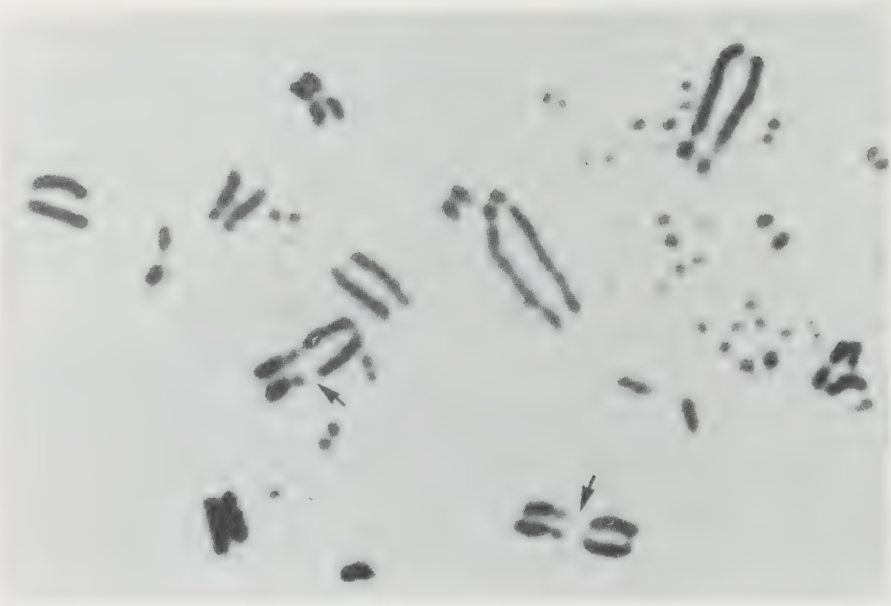


Figure 19.8. Chromosome gap on chromosome 2. Found only in embryos homozygous for a pericentric inversion on chromosome 1. The homozygous condition is lethal at 24-48 h incubation.

displaying a chromosome gap on chromosome 2 (Figure 19.8), not seen in chromosomally heterozygous or normal embryos. The embryos were obtained after 24 h incubation, the cells frozen in a 3:1 methanol:glacial acetic acid fixative, and stained with carbol fuchsin. More studies are needed to determine the basis for this phenomenon.

A pericentric inversion of chromosome 2, inv(2), induced by X-irradiation of semen (Wooster et al., 1977) has nearly normal reproductive performance. Studies of synaptonemal complexes (Kaelbling and Fechheimer, 1981, 1985) showed a high incidence of nonhomologous pairing and unpaired loops. With no recombination in the inverted segment, dp/df gametes are not produced. Gametes from heterozygous carriers will be genetically balanced, carrying either a structurally normal or an inv(2) chromosome. With no recombination between inverted and noninverted segments, the genetic content is locked in, precluding genetic variation. This may be advantageous, exerting a positive selection pressure, or disadvantageous, with genes exerting negative selection pressure. A disadvantageous gene, the recessive semilethal mutation shankless (*shl*), is associated with the inv(2) (Langhorst and Fechheimer, 1985). When the inversion is homozygous only *shl* progeny are produced. Hatchability is nearly normal, but *shl* birds are difficult to rear to reproductive age. A number of matings have failed to separate the two genetic markers, one cellular, inv(2), and the other morphological, *shl* (Fechheimer, 1983, personal communication; Bitgood and Shoffner, unpublished).

In some bird species the Z may be acrocentric, but more often it is metacentric (Beçak et al., 1971). Pericentric inversions may account for these different morphologies. Banding and other technologies may be useful in resolving the homology between these two predominant configurations.

Silversides et al. (1988) reported that the morphological difference seen in chromosome 4 of African and Pilgrim geese was probably due to a pericentric inversion. This supports the theory that Chinese and African geese were derived from the Asiatic swan goose with the same karyotype, while the Pilgrim and other western breeds were derived from the European greylag goose with the same ancestral configuration. With the banding procedures now available, this would be an area of interest for evolutionary studies.

HETEROPLOIDY

Heteroploidy is any deviation from normal chromosome number. Heteroploid chromosome numbers can be either euploid or aneuploid. Euploidy is an exact duplication of the basic monoploid (haploid) number of chromosomes characteristic of the species. Euploidy includes monoploidy, diploidy (the normal avian condition of two sets of chromosomes), or polyploidy (three or more sets of chromosomes). Aneuploidy is when one or more chromosomes of the euploid set are absent or present in addition to the euploid set (Rieger et al., 1976).

Most forms of heteroploidy are lethal during early embryonic development. Cytological examination of embryos after 24 h incubation identifies the majority of abnormal chromosomes complements that are lethal.

A frequency range for spontaneous abnormalities and their effect on viability was provided in cytological studies of embryos from chromosomally normal parents (Miller et al., 1971; Bloom, 1972). Chromosome aberrations found in various stocks ranged from 0.4-12.7 percent depending upon the strain, with a higher value found in a meat-type rapid growth line. A wide variety of aberrations has been identified, including haploids, polyploids, various combinations of haploid and polyploid mosaics, monosomics, trisomics, deletions, and spontaneous rearrangements (Bammi et al., 1966; Bloom and Buss, 1966; Bloom, 1970a, 1972; Miller et al., 1971; Lodge et al., 1974; Mong et al., 1974; Snyder et al., 1975; Dinkel et al., 1979; Shoffner et al., 1979).

Bloom (1972) noted differences when embryos from different strains and varieties of chickens were examined. An Araucana strain produced only haploids, whereas a junglefowl strain and an obese strain produced only triploids. Considerable variation existed among individual hens within strains, suggesting that ploidy may be under genetic control. Dinkel et al. (1978) studied three highly inbred lines. Two of these lines had no aberrant embryos (77 and 58 examined), whereas the third line had 3.0 percent (8/261).

Tetraploids or mosaics with tetraploid cell lines have been found in embryos (Miller et al., 1971; Bloom, 1972; Snyder et al., 1975). In all cases the chromosome complement of the polyploid embryo or cell line was an exact double of the diploid chromosome complement, even when the diploid line was aberrant (Miller et al., 1971). Bloom (1972) reported both 4A-ZZZZ and 4A-ZZWW embryos, and Miller et al. (1971) found ZZ/ZZZZ and ZW/ZZWW mosaics. These can be caused by a failure of cytokinesis, either during the first mitosis following fertilization producing wholly tetraploid embryos, or in a single cell of an early diploid blastomere producing a tetraploid cell line. There have been no reports of developmentally viable full tetraploids or individuals with tetraploid cell lines.

In vivo ageing of sperm may lead to subsequent aberrations in the developing embryo. Lodge et al. (1974) observed that eggs collected 1-3 days after insemination had a .45 percent aberration rate, compared to a 2.22 percent aberration rate in eggs collected from these same matings 9-14 days after insemination. There was no difference between two strains of White Leghorn and a Columbian stock.

Chromosome abnormalities are commonly associated with early abortion. In mice and humans, a variety of chromosomal aberrants survive embryonic development resulting in abnormal individuals or individuals with reduced fertility. Birds have not been as intensively studied, so there are only a few reports of viable birds carrying unbalanced genomes. However, no phenotypic abnormalities have been noted. Shoffner et al. (1979) reported a ten-month-old F_2 progeny from a Ross's x emperor goose cross that was trisomic for chromosome 1, with one Ross's and two emperor chromosome 1s. A phenotypically normal chicken, trisomic for a microchromosome, was found by Bloom et al. (1978). Viable progeny that are disomic, trisomic and tetrasomic for this microchromosome are produced when trisomic parents are mated (Bloom and Bacon, 1985).

Thorne et al. (1987) reported viable mosaic chickens that were haploid-diploid, diploid-triploid, and haploid-diploid-triploid. Fertility of the diploid-triploid birds varied with the sex chromosome complement and the ratio of diploid to triploid cells. Fertility of the haploid-diploid birds was normal.

Many cases of viable triploid chickens have been reported, all of either genotype 3A-ZZW or 3A-ZZZ. Finding early embryos of 3A-ZWW genotype suggested that this sex chromosome combination is incompatible with development (see Miller et al., 1971; Mong et al., 1974; Wang and Shoffner, 1980 for reviews). However Mayr and Auer (1988) found a turkey that was 2A-ZWW. A more critical search for viable ZWW individuals is needed.

The majority (at least 80 percent) of triploids in chickens originate from faulty disjunction during oogenesis. In a summary paper, Mong et al. (1974) reported that 22 of 54 triploid embryos were 3A-ZWW. This can only occur by nondisjunction in meiosis II during oogenesis. An approximately equal number (25) of 3A-ZZZ embryos were produced as expected from this same type of failure. Seven other embryos were 3A-ZZW and could have resulted from faulty oogenesis in meiosis I, diploid sperm, or polyspermy. Snyder et al. (1975) found eight triploids whose chromosome complement agreed with these conclusions.

Four 3A-ZWW triploids from diploid ova (WW) were found during the initial four weeks of egg production (Miller et al., 1971). When egg production begins pullets ovulate at irregular intervals. This may disrupt the reduction division cycle, resulting in diploid gametes (Mong et al., 1974). After the laying cycle stabilizes, the incidence of triploidy decreases. These same authors found no evidence of clustering of triploids within dams. Snyder et al. (1975) found an influence of dams in a broiler and an egg production strain in the frequency of haploid and haploid/euploid mosaic embryos. The broiler strain had a higher incidence of triploid embryos. They found no difference between sire lines.

Wang and Shoffner (1980) postulated that triploids could originate either by failure of the first or the second meiotic division during oogenesis. They found some triploids which could have arisen from suppression of the first meiotic division, and others which could have arisen from suppression of the second meiotic division.

Zartman and Smith (1975) mated males homozygous for a chromosome rearrangement to females carrying normal chromosomes. Two triploids and one haploid/triploid mosaic were found in 67 embryos. The triploids were derived from 2A-ZW diploid ova. In the mosaic, the haploid cell line was paternal in origin, but the triploid cell line was derived from a 2A-ZW diploid ovum. The diploid ovum could have resulted from either a failure of the reductional division or a reunion of the first polar body with the egg pronucleus at some point prior to the first cleavage division of the zygote. A less likely event would be the fusion of two ova.

Fechheimer and Jaap (1978) used a similar mating scheme to mark the paternal chromosome contribution. All 2n embryos recovered were heterozygous for the rearrangement, showing normal segregation and fertilization. All haploid embryos and those with haploid cell lines had a paternal rearranged chromosome. Nine triploids and the triploid cell line in a 1n/3n mosaic had one rearranged chromosome, indicating the 3n cells arose from a 2n ovum fertilized by a 1n sperm. One tetraploid and three 2n/4n mosaics were found. The 4n cells contained two rearranged chromosomes, suggesting a failure of cytokinesis in an early embryonic cleavage division. Two 2n/3n mosaics were found. One had sex chromosomes of ZW/ZZW and one translocation chromosome in each cell line, suggesting failure of cytokinesis. The other was ZZ/ZZZ with a single rearranged chromosome in the 2n line, but two rearranged chromosomes in the 3n line, indicating two paternal chromosome sets. These authors noted a clustering of triploid embryos from certain hens, but did not comment on a possible genetic influence.

Dinkel et al. (1979) studied disjunction types in four translocations, and found no differences between sires or dams in frequencies of types of segregations. A difference was found in transmission of complementary products within some heterozygous dams, and they suggested anaphase lagging as the cause.

One instance of heteroploidy was useful in clarifying the morphology of a pericentric inversion in chromosome 1 in the chicken (Bitgood et al., 1982). An embryo was recovered (Figure 19.9) that in effect was trisomic for chromosome 1. The dam was chromosomally normal and provided a normal chromosome 1 to the zygote. The sire was heterozygous for a pericentric inversion, and as a result of crossing-over within the inversion loop produced complementary dp/df chromosomes, one deficient for a segment of the short arm, the other chromosome duplicated for the same segment. These dp/df chromosomes underwent nondisjunction during meiosis, and upon fertilization a 2n+1 zygote was created. This embryo contained a normal 1, a duplication 1 and a deficient 1 in all cells examined. The distinct morphological changes in the two arms when compared to the normal 1 permitted description of the recombinant chromosomes.

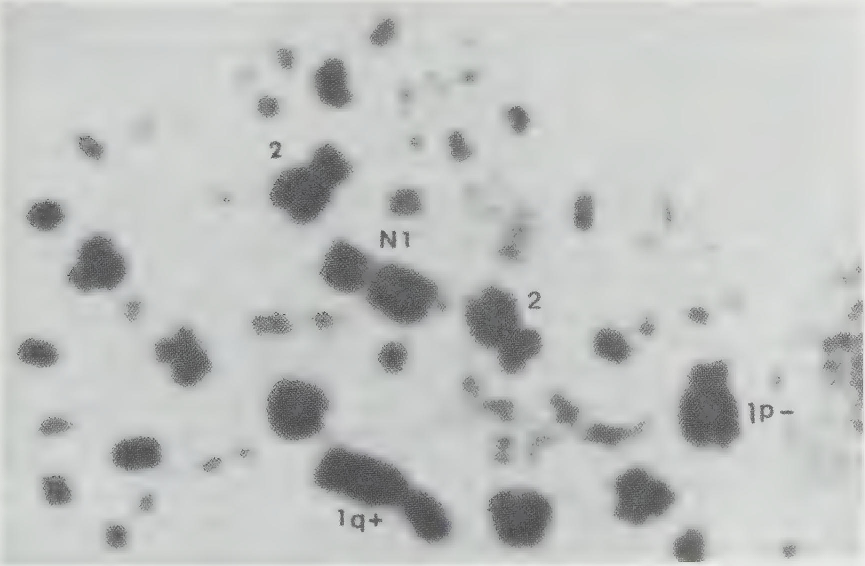


Figure 19.9. An embryo with three different structural arrangements of chromosome 1. A normal, a 1q+, and a 1p- chromosome are all present in the same cell, resulting in an apparent 3n amount of chromosome 1, but structurally different from a trisomic.

For more detailed reviews on heteroploidy in chickens, see Fechheimer and Jaap (1980), and Fechheimer (1981).

HYBRIDS

Gray (1958) compiled a sizable list of reported interspecific and intergeneric bird hybrids, but very little is known about their genetic homology. It is commonly believed that the incompatibility in development and reproduction of interspecific and intergeneric hybrids is due to chromosomal differences of the parental species. Only a few cases have been investigated cytogenetically (see Shoffner, 1974 for a review). Some hybrids such as the muscovy x Pekin mule duck, while sterile, are developmentally compatible and commercially important in some areas of the world. Mott et al. (1968) found that the long arm of the muscovy chromosome 1 was longer than that of its counterpart in the Pekin when the hybrid was studied. The muscovy Z chromosome was acrocentric, whereas the Pekin Z chromosome was subacrocentric. The difference in morphology of these two chromosomes was postulated to account for the sterility of the hybrid.

Haldane's rule states that if one sex is absent, rare or sterile in crosses between species, it is the heterogametic sex (Rieger et al., 1976). This seems to apply to avian crosses. The disadvantage of the embryonic heterogametic female could be due to a deleterious mutation(s) on the W, normally compensated for by a locus on either the Z or an autosome. In hybrids the genetic composition of the opposite parent may not offset the effects of the undesirable gene. The direction of the cross appears to influence hybrid viability. No studies have been conducted on paternal Z chromosome/maternal mitochondrial compatibility.

Painter and Cole (1943) mated pigeon males to dove females. All the adult F_1 birds were both genetically and phenotypically male. They theorized that female embryos died during development. The reciprocal cross yielded viable female and male progeny. When a chicken male and Japanese quail females were mated, all hatched chicks were male (Bammi et al., 1966). The ZW females died early in embryonic development.

Harada and Buss (1981b) crossed chicken males with turkey females. Both male and female embryos were found at early stages, but female embryos did not survive to hatch time. With the reciprocal cross, there apparently was no evidence of hybridization, but cell division was initiated in all chicken eggs. The authors hypothesized that the turkey sperm stimulated parthenogenetic development. It is also possible that fertilization occurred but the embryos died shortly after this.

Shoffner et al. (1979) recovered hybrids of Ross's and emperor geese. The F_1 birds were fertile, even though morphological differences existed in chromosome pairs 1 and 3. The authors suggested that a translocation occurred between these chromosomes during evolution. Silversides et al. (1988) crossed African and Pilgrim geese. A difference in morphology of chromosome 4 was found, apparently due to a pericentric inversion.

Greenfield et al. (1986) studied heterochromatin distribution in chicken x Japanese quail hybrids. They reviewed literature describing the chicken heterochromatin as dispersed in small chromocentres distributed uniformly in the cell. The Japanese quail chromatin aggregates in large central masses associated with the nucleolar RNA. This is useful for distinguishing parentage of cells in quail-chick chimeras. In hybrid cells, there are one or more large central chromatin masses, with dispersed chromatin in a few chromocentres.

PARTHENOGENESIS

Parthenogenetic development has been documented in chickens and turkeys, and parthenogenetic turkey males have become successful sires. Olsen (1965) reviewed the results of a long-range project with turkeys in which 45 percent of all infertile eggs from the selected strain showed some development. Twenty-five percent of hatched poults survived to maturity, and some produced viable sperm with resultant progeny. Over the period of the study, more than 25 sired progeny. Injection of parent hens with live vaccine against fowl pox, Newcastle disease, and Rous sarcoma increased the incidence of development in eggs from chickens and turkey parthenogenetic strains (Olsen, 1956; 1962). Killed-virus vaccines were not effective.

Olsen (1966b) and Olsen and Buss (1972) attempted to determine how diploid embryos developed from unfertilized ova, by examining segregation of down color in parthenogenetic poults from heterozygote dams. They speculated that the one heterozygous parthenote resulted from suppression or re-entry of the second polar body. However, Harada and Buss (1981a) proved that turkey parthenotes originated as haploids. Diploidy occurs later, probably as a result of cell fusion or inhibition of cell division.

Olsen et al. (1968) suggested that in chickens this may be under the control of a single autosomal recessive locus. One strain of Dark Cornish showed 6.38 percent parthenogenesis. Other Cornish strains showed a lower propensity (Olsen, 1966a), whereas other lines of chickens showed very little parthenogenesis.

Harada and Buss (1981b) found that when a turkey male was crossed with a chicken hen, there was no evidence of hybridization, but cell division was initiated in all chicken eggs. They hypothesized that turkey sperm had some stimulating effect on parthenogenetic development in chicken eggs.

TECHNIQUES IN AVIAN CYTOLOGY

Methods For Obtaining Cells

Birds have some advantages over other species for cytogenetic research. In addition to standard blood and tissue culture techniques, *in vivo* feather pulp and early embryos are available for study. Each technique has its advantages and disadvantages; one may be more useful in one laboratory, while another is better suited for use in a different laboratory.

Feather pulp. This procedure was first described by Sandnes (1954) and refined by Shoffner et al. (1967). It takes advantage of the high mitotic rate in rapidly growing pulpy tissue found at the base of pin feathers. The bird is injected with a mitotic inhibitor such as colchicine and the feather pulp is harvested two hours later. After a brief hypotonic treatment the pulp is placed in a fixative. Squash preparations are examined with a phase microscope. This technique is useful for rapid screening of individuals for chromosome complement. It is not recommended for birds less than 5-6 weeks of age that are potential breeders. If birds lacking pin feathers need to be examined, mature feathers can be pulled from a small area and pin feathers should appear in 7-10 days. The neck region is a productive area to use.

Embryos. The availability of embryos for cytogenetic study makes the bird an excellent model. Planned matings can be established to provide embryos on a daily basis. Embryos can be harvested at practically any stage of incubation for chromosome studies. Embryos up to 24-48 h incubation are used to demonstrate unbalanced gametes (Shoffner et al., 1967; Lodge et al., 1973). An injection of colchicine 45-120 min prior to harvest generates a high number of C-metaphases. These are the metaphase configurations accumulated by temporary blockage of spindle fibre formation by colchicine or other mitotic inhibitors. Two hours of further incubation is needed for early embryos which have not yet formed blood vessels, whereas 45 min is adequate if blood vessels are present.

Leukocyte culture. These cultures are used as in other species (Shoffner et al., 1967). A slightly higher incubation temperature is necessary compared to mammalian cultures (41° C vs 37.5° C). Cultures are grown for 48-72 h. A small amount of mitotic inhibitor is added and the culture is incubated for another 45 min. The cells are treated with a hypotonic solution and placed in a fixative. Either an air-dried or a squash technique is used to prepare slides.

Cell culture. Cell cultures are used in many laboratories. A number of cell lines are available but they have not been routinely used for avian cytogenetic studies, probably because the embryo is a natural tissue culture easily obtained and used for chromosome studies.

Meiotic studies. Diakinesis configurations (Figure 19.2) are obtained from sexually mature males by biopsy of a testis or the male can be sacrificed for a testis sample. Tissue pieces 1-3 mm in diameter are treated in hypotonic solution, then transferred into a fixative. Tissue can be prepared for use by either the air-dried or squash procedures.

Study of female meiosis is difficult as ova develop to the diplotene stage prior to the chick hatching, and are held at this stage until just prior to ovulation in the adult hen. Intermediate stages occur relatively rapidly and are difficult to capture although Solari (1977) successfully used whole mount spreading of oocytes from newly hatched female chicks to study SC formation.

Techniques For Cytochemical Differentiation (Banding)

Banding techniques are useful for marking specific regions of chromosomes, as well as for differentiating between chromosomes that are morphologically similar. These procedures have identified break/reunion points of chromosome rearrangements. Comparative chromosome banding is used to study phylogenetic relationships. Reviews on cytogenetic methods including the banding techniques have been published by Pathak (1979), Sumner (1982), Fechheimer (1985), and Auer et al. (1987). Hsu (1973) reviewed the banding procedures in use in the early 1970s. Sequential application of different procedures to the same chromosome sets will give additional information about chromosome structure.

C-banding. C-bands are generated by treatment with an alkali followed by a saline/sodium citrate treatment (Pardue and Gall, 1970). The procedure differentially marks constitutive heterochromatin (C=constitutive), which includes satellite or highly repetitive DNA. In the chicken this is confined largely to the W chromosome, the terminal end of the long arm of the Z chromosome, and small regions on some of the microchromosomes. Pollock and Fechheimer (1981) reported two C-band polymorphisms on the chicken Z chromosome. In avian species the W chromosome is C-band positive and this is useful in determining sex chromosome complement.

Q-banding. This was the first banding technique described (Caspersson et al., 1968). The bands are created by staining with quinacrine mustard (Q=quinacrine) or other fluorochromes (fluorescing dyes) such as acridine orange. A fluorescent microscope is required to view and/or photograph the chromosomes. Because of the extra equipment and expense as well as a tendency for Q-bands to overlap, most banding studies utilize G-banding, which is similar and more definitive.

G-banding. A variety of treatments will produce these bands, although the trypsin method is the most common procedure in use for analysis of avian chromosomes. The procedure generally calls for Giemsa stain (hence G-bands), but other stains such as Wright stain have been used. This procedure was used by Bitgood et al. (1981) to identify the break-reunion points of a rare cyclical translocation in the chicken, apparently the first in vertebrates.

The number of bands on a chromosome will vary depending on the stage of contraction of the chromosome. Elongated chromosomes, for instance in premetaphase, will display more bands than midmetaphase chromosomes. The positive staining G-band dark and Q-band bright bands are believed to mark regions rich in guanine and cytosine that replicate DNA early in the S phase (synthesis phase when DNA is replicated) of the cell cycle. The G-band nonstaining (light) and Q-band nonstaining (dark) bands contain regions rich in adenine and thymine and are late replicating.

A proposed G-band map of the chicken is shown in Figure 19.10. Landmark regions were designated by an *ad hoc* committee that met in St. Paul, MN in June, 1980 (St. Paul Conference on Standardization of Avian Chromosomes, 1980, unpublished). Members of this committee were B.M. Biederman, J.J. Bitgood, S.E. Bloom, N.S. Fechheimer, C.C. Lin, J.S. Otis, and R.N. Shoffner, chair.

R-banding. These bands are the reverse of Q- and G-bands (R=reverse), first described by Dutrillaux and Lejeune (1971). This procedure uses a regular light microscope. The mechanism of creation of R-bands is not clear. It does appear that this procedure results in the least extraction of proteins from the chromosome complex. Another procedure calls for addition of 5-bromodeoxyuridine (an analog of uridine) in the cell culture medium and use of a fluorescent stain. This procedure gives similar but not the same results as R-banding (Pathak, 1979). When tips of chromosomes contain a light staining G-band, the R-band procedure is useful if telomeres are being studied, or chromosome measurements are being taken.

Telomere (kinetochore) staining. A modification of the R-banding procedure will produce T-bands (stained telomeres only) (Dutrillaux, 1973). There are several other procedures that will stain kinetochores (centromeres), but the mechanism is not understood.

Ag-NOR staining. This procedure, designated as the Ag-AS procedure (Goodpasture and Bloom, 1975), uses silver nitrate cytochemistry to stain the nucleolar organizing regions (NORs) on chromosomes. The NORs are the regions on the chromosomes that contain the ribosomal RNA (rRNA) repeated gene family that encodes the 18S and the 28S rRNAs. The Ag-AS technique stains a nucleolar protein (or proteins) believed to be associated with the transcription complex (Hernandez-Verdun, 1983).

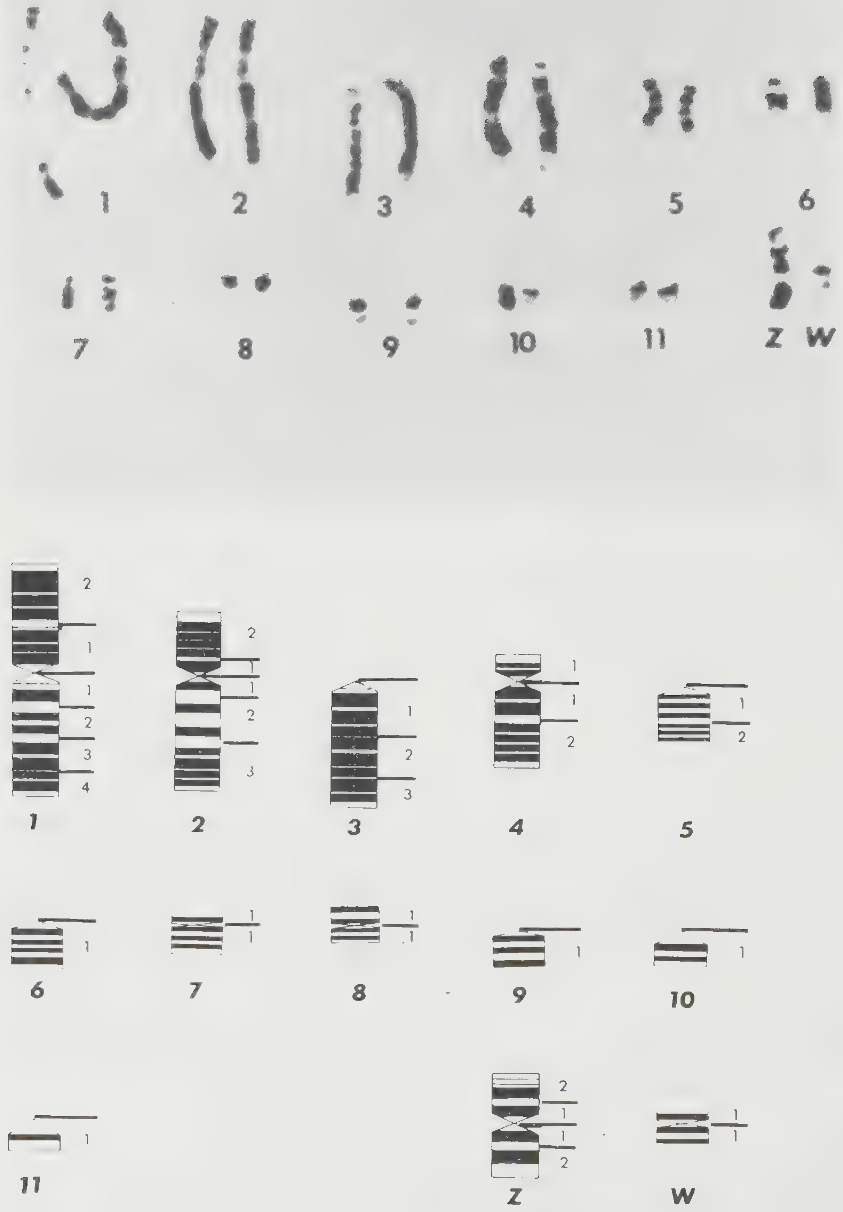


Figure 19.10. A G-band karyotype (upper) and a proposed G-band map (lower) of the chicken. A composite of work by G.A. Opitz and J.J. Bitgood.

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Chapter 20. PLEIOTROPIC AND ASSOCIATED EFFECTS OF MAJOR GENES

P. Mérat

MAJOR OR MARKER GENES — PLEIOTROPY OR LINKAGE

Marker genes identified by qualitative criteria (visual, biochemical or serological) may show association with quantitative traits either because of *pleiotropy*, defined by Johansson and Rendel (1968) as the case when a gene influences two or more traits, or because of *linkage* with other genes. It is very difficult to discriminate between a pleiotropic effect and close linkage, except when a reasonable interpretation of the quantitative effect of the marker gene may be derived from its primary effect.

Indications for discriminating between pleiotropy and/or close linkage and loose linkage are the persistency of associated effects across generations of the same population and across different populations. A pleiotropic effect is consistently associated with a gene, while an effect associated through linkage will be reversed in some genetic groups. However possible interactions of the relevant gene with the remainder of the genome or with the environment cannot be ruled out as summarized in Table 20.1.

Another approach for single genes with large effect on quantitative traits consists of detecting them directly from examination of statistical parameters, for instance from existence of several modes or of deviations from normality in frequency distributions, and heterogeneity of variances according to parental genotypes for the supposed major gene(s). Several methods have been proposed (see review by Hanset, 1982). These methods are only poorly discriminant, and even if genes with large effect are suggested, the problem of identifying genotypes is generally unsolved. In domestic birds their application has not yet had significant results.

In reviewing quantitative effects associated with marker genes identified by various criteria, an attempt will be made in each case to draw together all the effects on economic value. Most published work has concerned domestic fowl. The limited research on other species is reviewed at the end of this chapter.

Table 20.1. Discrimination between pleiotropy and linkage.

Constancy of effects	Hypothesis
Constant across generations	Pleiotropy or tight linkage
Variable (decreasing) in successive generations:	
- genetically constant population	Linkage
- genetically varying population	Linkage or pleiotropy with gene x genome interaction
Constant among various populations	Pleiotropy
Variable between populations	Linkage or pleiotropy with gene x genome interaction
Variable between environments	Gene x environment interaction

GENES REDUCING BODY SIZE: THE SEX-LINKED DWARFING GENE (*dw*)

The sex-linked dwarfing gene was described by Hutt (1959). It has appeared by mutation in diverse populations (Guillaume, 1976). As shown by Scanes et al. (1983) and Decuypere et al. (1986), the *dw* gene lowers plasma triiodo-thyronine (T3) and IGF I, and it slightly increases thyroxine (T4) and growth hormone (GH). Deiodination in the liver is reduced in spite of the presence of deiodinase in the hepatic cells (Decuypere et al., 1986), and the binding of growth hormone to hepatic cells is lowered in dwarf birds (Leung et al., 1987). These changes help to interpret the depressing effect of the dwarfing gene on growth rate, owing to the influence of T3, GH and IGF I on it (Scanes et al., 1984). The consequences on egg production are less obvious.

Effects Of The *dw* Gene On Economic Performance

For access to the extensive literature on this subject, refer to the reviews by Guillaume (1976), Polkinghorne (1973), and Horst and Petersen (1976) concerning egg production, and by Ricard (1976) and Mérat (1984) dealing with broiler production.

Body weight and body composition. The *dw* gene has no appreciable effect on size of the day-old chick but it delays the resorption of yolk (Delpech, 1968; Strong and Jaap, 1977). A progressive reduction of body size takes place posthatching in comparison with a normal-sized bird of the same origin, approximating 30 percent in females and 40 percent in males from 8-10 weeks of age (Guillaume, 1976; Ricard, 1976). Several studies (see Mérat, 1984) showed that body weight is less depressed by the dwarfing gene in a line selected for high body weight than in a line selected in the opposite direction. However, this does not appear in all comparisons between heavy and light populations (Table 20.2).

Dwarf chicks are fatter than their normal counterparts, but conversely, in the mature hen the percentage of abdominal fat is decreased by the *dw* allele (Ricard, 1976) possibly owing to a higher lipid output for egg production in proportion to body size.

Table 20.2. Effects of the *dw* gene (percent deviation from Dw^+) in females in laying or broiler-type stocks.

Trait	Light layers (including Leghorns)				Medium-sized layers				Broiler type			
	n[1]	min	max	median	n	min	max	median	n	min	max	median
Body weight	15	-39	-17	-32	12	-36	-24	-31	6	-35	-23	-31
Egg number (hen-day)	24	-22[2]	-4	-14	14	-19	+1	-7	6	-7	+21	+3
Mean egg wt.	23	-12	-3	-8	14	-11	-4	-8	4	-4	-2	-3
Feed efficiency[3]	16	-8	+34	+3	10	+8	+22	+13	5	+18	+57	+37

[1] Number of reports reviewed from available literature.

[2] Or -53 if including reports of unusual delay in sexual maturity of dwarfs (see Guillaume, 1976).

[3] Per g egg for layers; per dozen eggs in broiler types.

Egg production traits. Some effects on egg production traits differ in layer-type lines (Leghorn or medium-sized brown egg type) or in broiler lines (Table 20.2).

The mean age at first egg is retarded by a few days to two weeks. Egg weight is reduced in connection with lower body weight, but less on the average in broiler strains than in layer strains. Egg number is decreased in layer-type stocks, and more in the Leghorn light type than in the medium-sized type, but not in broiler strains. As a consequence, feed efficiency of broiler breeder females is improved by about 25 percent (per gram of egg) by the dwarfing gene, since egg mass is only affected by the moderate decrease of mean egg weight. In medium-sized laying stocks, in spite of the depressing effect on egg number, the feed efficiency of *dw* hens compared with normal females shows a mean gain of around 13 percent, and in Leghorns feed efficiency is more frequently improved than worsened.

Abnormal eggs (double-yolked or soft-shelled) are almost suppressed by the *dw* gene (see Guillaume, 1976, and Mérat, 1984). In connection with this is the absence of an unfavorable effect of the *dw* gene on laying rate in heavy strains. Jaap and Clancy (1968) have shown that broiler hens have an excessive number of large growing follicles causing a high percentage of abnormal eggs and a depression of laying rate, while laying-type hens have a lower number of large follicles more compatible with a normal laying rate. In both cases, the sex-linked *dw* gene decreases the number of large follicles; in broiler strains it shifts it towards a normal value suppressing abnormal eggs, while in laying-type populations it lowers this number below the value corresponding to maximum egg production.

Another effect of *dw* is a shortening of clutch length (Wood, 1971; Amin-Bakhche and Mérat, 1975; Yoo et al., 1984) associated with a longer time interval between successive ovipositions in comparison with the Dw^+ gene.

It has been shown by Rapp (1970) and others (see Mérat, 1984) that the *dw* gene reduces (generally by more than half) the percentage of cracked eggs in batteries, in light as well as in heavy strains. It has no appreciable effect on egg shell thickness or egg specific gravity. The reduction of egg breakage by *dw* may be due to lower body weight of the hen, since any weight applied to the cage floor increases the mechanical insult caused to the laid egg (Carter, 1975). In addition the height of drop of the egg may be less because of the shorter shanks of dwarf females. The reputedly quiet temperament of *dw* hens may also play a role.

No consistent effect of the *dw* gene on Haugh units has been found. Demarne et al. (1984) showed that in triglycerides of the yolk, the percentage of linoleic acid is changed from a mean value of 14.8 percent in Dw^+ females to 22.9 percent in *dw* hens while the corresponding figures for oleic acid are 51.8 and 43.3 respectively.

Mortality. For overall mortality, the majority of results show nonsignificant differences between Dw^+ and *dw* hens; the advantage seems to be more often in favor of dwarf birds than the reverse. Quisenberry (1972, cited by Guillaume, 1976) comparing dwarf and normal hens at four different protein levels (from 22-14 percent) observed that dwarf layers had a better survival rate with the highest protein level and poorer with the lowest.

Concerning specific diseases, Table 20.3 summarizes recorded effects associated with the *dw* gene in comparison with the normal genotype.

Fertility and hatchability. Fertility was not found significantly different for Dw^+ and *dw* breeding females mated to similar males, for both layer and broiler lines (see reviews by Guillaume, 1976 and Mérat, 1984).

Table 20.3. Effect of *dw* on response to specific diseases and on immune response.

Trait	Effect of *dw* compared to normal genotype	Reference
RESPONSE TO DISEASE		
Marek's disease	More resistant	Rapp (1970) Meurier and Mérat (1972)[1] Hartmann (1978)[1]
Newcastle disease	No difference	Stephens & Dreyfuss (1978)[1]
Coccidioses, Salmonelloses, *E. coli* infection, Mycoplasmoses	No difference	Haas et al. (1975)[1]
E. coli infection	More susceptible in a low-weight line, not in a high-weight line	Reddy et al. (1975)[1]
Spirochetosis	More resistant (smaller red cells more rapidly destroyed?)	Joshi et al. (1980)
Laying accidents	Less susceptible	Rapp (1970)
Rickets	More susceptible	Austic et al. (1977)
Dermatitis of nutritional origin	More susceptible	Roush & Arscott (1983), Burger et al. (1984)
IMMUNE RESPONSE; ANTIBODY PRODUCTION		
Antibodies against *E. coli* and SRBC	Lower titres in a light line, higher titres in a heavy line	Mauldin et al. (1978)[1] Marsteller et al. (1980)[1]
Antibody production at high temperature	Higher antibody production and less mortality	Kassim et al. (1982)

[1] Reference citations in Mérat (1984).

In heavy stocks a slight advantage for dwarf females seems to exist for hatchability (Ricard, 1976, and reviews by Guillaume, 1976 and Mérat, 1984) likely because of fewer abnormal eggs.

Specific Responses Of Dwarf Birds To Environmental Factors

Table 20.4 summarizes reported cases of a differential response associated with the *dw* gene to environmental factors. For some of them there are hints of a physiological basis. More protein catabolism with increased gluconeogenesis was suggested by Guillaume (1976); Jones et al. (1986) conclude similarly. In addition the egg mass output in relation to body weight is higher in minilayers. Fat metabolism is affected by the dwarfing gene, with reduced lipolysis (Guillaume, 1976) and less fatty acid synthesis is likely (Burghelle-Mayeur, 1988). Concerning calcium metabolism in layers, proportionally more calcium

Table 20.4. Differential response to environmental factors associated with the *dw* gene.

Nature of the effect	Reference
PROTEIN CONTENT OF THE RATION	
dw hens more responsive in egg weight and/or laying rate to increased protein or methionine	Arscott and Bernier (1968, 1970)[1] Quisenberry (1972)[1] Cherry and Siegel (1978)[2] Bell et al. (1983)
Other results with no gene x feed interaction	Magruder and Coone (1969)[1] Cherry and Siegel (1979)[2] Sadjadi et al. (1983a) Guillaume (1973)[1]
ENERGY AND LIPIDS	
Energy level not generally affecting the dwarf layer	Guillaume (1976)
Slight effect of more lipids in feed on laying rate of *dw* hens	El Attar et al. (1986)
MINERALS	
Susceptibility to rickets	Austic et al. (1977)
Light *dw* layers affected by low calcium in feed; separate calcium feeding beneficial (possibly more than for normal hens?)	Arscott et al. (1962)[1] Khoo (1977); Bordas and Mérat (1986)
LIGHT	
Slight relative advantage of *dw* layers (medium-size) on 26 or 27 h daylight cycles	Mérat and Bordas (1986) Mérat et al. (1988)
TEMPERATURE	
Decreased feed consumption at low ambient temperatures	Rajaratnam et al. (1969)[1]; Rapp (1970)
No decrease at 18°C compared to 25°C	Mérat (unpublished)
Less depression of egg number and mean egg weight at high temperature than for Dw^+ hens	Mérat et al. (1974)[2]; Horst and Petersen (1979)[2]; Becker (1983)
Other cases with no relative advantage of *dw* layers in hot conditions	Ahmad et al. (1971)[1] Wilson et al. (1972) Bullermann and Hiendl (1981)[2] Selmi & Horst (1984)
FLOOR OR CAGE HOUSING; BIRD DENSITY	
No interaction with floor vs cage keeping	McClung et al. (1971)[1] Strain and Piloski (1976)[2] Lin et al. (1979) Trefil et al. (1987)
No interaction with bird density	Dorminey et al. (1972)[1]

[1] Reference citations in Guillaume (1976).

[2] Reference citations in Mérat (1984).

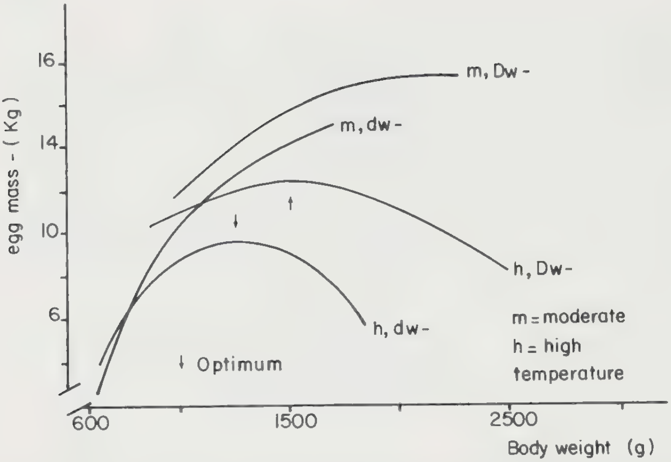

Figure 20.1. Relation between egg mass and body weight according to genotype at the *Dw* locus and ambient temperature. (From Horst and Petersen, 1979).

appears to be drawn from the feed by the *dw* hen during shell formation (Bordas and Mérat, 1976). There is no reason to suspect a specific response of *dw* hens to light. However, as they show an increased time interval between successive ovipositions (Amin-Bakhche and Mérat, 1975; Yoo et al., 1984) they may benefit from light cycles longer than 24 h, avoiding the between-clutch intervals caused by lag between the hen's internal cycle and the 24 h cycle. Finally, for response to heat it is known that at high temperatures a small body size allows faster heat dissipation because of increased body surface in proportion to its volume. But things may not be so simple with the *dw* gene; in addition to results in Table 20.4, Figure 20.1 from Horst and Petersen (1979) suggests a different optimal body weight for the Dw^+ and *dw* genotypes according to ambient temperature.

Interaction With The Genetic Background And Utilization Of The *dw* Gene For Egg Production

In layer populations, potential interest in the dwarfing gene lies in better feed conversion during egg production, a 25-30 percent saving on feed consumption up to sexual maturity (Durham, 1972), fewer cracked and soft-shelled eggs, a 20 percent lower volume of droppings (Gyles, 1973), and less space requirement per laying hen. Restricted feeding programs are avoided in medium-sized stocks. The increased proportion of polyunsaturated fatty acids in the eggs (Demarne et al., 1984) may be slightly favorable for human nutrition. Opposed to these advantages is the lowering of mean egg weight and in general of egg number in light strains. It is therefore necessary to evaluate conditions optimizing the total balance.

Optimal body weight. Table 20.2 shows that the dwarfing gene is more beneficial on average in brown-egg type populations than in lighter Leghorns. Further for each type Figures 20.2a and 20.2b summarize from the literature the average adult body weight of a population plotted against the percent deviation caused by the *dw* gene on egg number and mean egg weight.

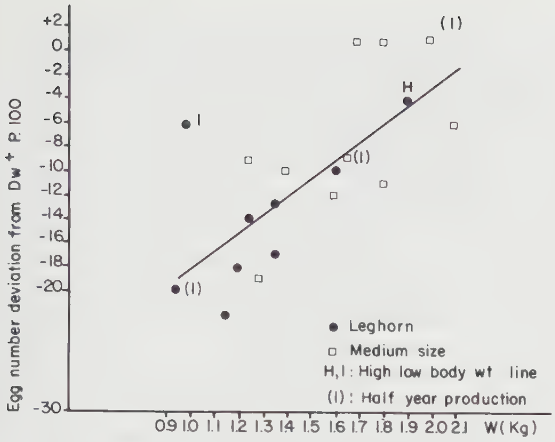

Figure 20.2a. Effect of the *dw* gene on egg number in layer-type lines according to body weight (W) of *dw* females. (From 19 research reports).

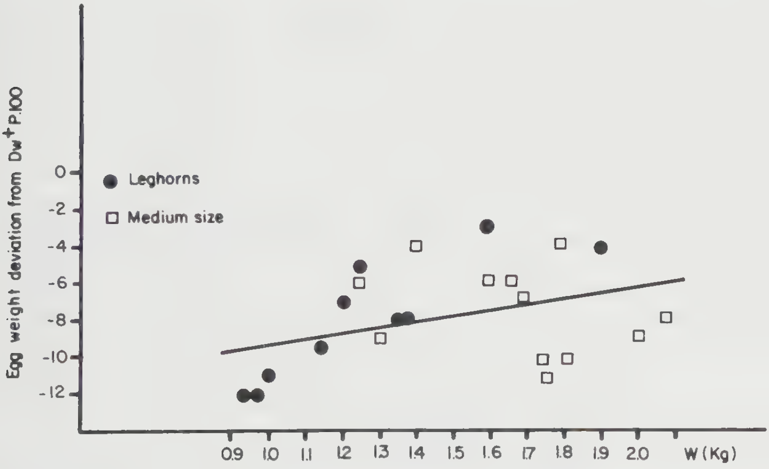

Figure 20.2b. Effect of the *dw* gene on mean egg weight according to body weight (W) of *dw* females. (From 19 research reports).

In spite of heterogeneity of the data, including several sources of mutation, they do not deviate from a unique linear regression; the lower the mean body weight, the larger the depressing effect of the *dw* gene on egg number and the less favorable is its effect on feed efficiency. On average, this latter effect begins to be positive when adult *dw* hens exceed one kg. This seems to be in agreement with the observation of a positive phenotypic correlation within *dw* laying-type strains between egg number and body weight (see Mérat, 1984). These results meet the suggestion by Horst (1982) and Bonitz (1986) that a sufficient body size is needed for minilayers.

Genetic improvement of egg production. Table 20.2 shows that there are populations, including Leghorns, where the effect of the *dw* gene is more beneficial than the average, and the advantage of the *dw* gene can be increased by long-term selection for egg production (Ngam 1987). The results of Yoo et al. (1984) however suggest combining the dwarf gene with selection for a short time interval between ovipositions. Leenstra et al. (1986) suggest introducing *dw* into a line producing a high percentage of multiple-yolked eggs; as in heavy strains, the *dw* gene in this background would shift the ovulation rate closer to an optimal value, success depending however on the absence of unfavorable correlated responses to selection for multiple yolks.

The disadvantages associated with the *dw* gene may be minimized by heterosis as in a Leghorn x Australorp cross (Yoo et al., 1980 cited by Horst, 1981). If this is general the prospect for the dwarf gene in egg production stocks may be more optimistic than predicted from results in pure lines.

Avoidance of underfeeding for dwarf layers. In addition to poor protein utilization by dwarf birds, protein intake is expected to be lower for smaller hens relative to their production (Nesheim, 1975). Hence, a tendency to underfeeding is suggested for dwarf birds. Dwarf chicks take more time to ingest less feed (El Attar et al., 1983). Dwarf birds decrease their feed intake at low ambient temperatures (Rapp, 1970; see also Guillaume, 1976). The naked neck gene, which increases feed consumption, has a beneficial effect on the laying rate of dwarf layers (Mérat, unpublished). Furthermore, a subnormal food and protein intake affects clutch length (Moreno et al., 1970) in a way similar to the effect of the *dw* gene in laying stocks. The opinion that increasing the feed intake of dwarf layers might improve their production was put forward by Rapp (1970), Durham (1972), Horst and Petersen (1976), Bonitz (1986), and others.

Particular situations. Maximizing the advantages and/or minimizing the drawbacks of the *dw* gene may involve particular situations, for instance where egg size is of less or no importance as in eggs for further processing, or use of a dwarf dam line in an egg-production cross (Yoo, 1979, personal communication), or where savings on feed costs are the most important as in developing countries (Selvarajah et al., 1970 as cited by Guillaume, 1976).

The use of the sex-linked dwarfing gene for egg production should be compared with lowering body weight by conventional selection (French and Nordskog, 1973 as cited by Guillaume, 1976). A large part of the effects associated with the *dw* gene involve size reduction alone. However, there still may be possible unfavorable correlated responses in long-term selection for light body weight.

Utilization Of The *dw* Gene For Broiler Production

The reduction of adult body weight by the sex-linked dwarfing gene and its recessiveness allow production of normal-sized broilers from dwarf dams mated to normal-sized males. The feed consumption of the breeding female is reduced both in the rearing period and in the reproduction period (about 25 percent per g egg, more per dozen eggs). The effects on egg laying, hatching rate, and viability in heavy stocks are rather favorable, and on average four percent more chicks per dam can be expected, probably because of very few abnormal and cracked eggs (which would be especially important in batteries). The reduction of required space per hen may reach 40 percent. The fertility of smaller hens with heavy males in natural matings is not considered to be a major problem, although it may be impaired if the size disproportion becomes too high. The improved heat tolerance noticed for dwarf hens may particularly concern heavy stocks. These advantages are to be compared with the slight negative effect on growth of broilers in the terminal cross. The dwarf dam produces eggs lighter by 3-4 g slightly depressing initial growth of the chick. In summarizing 14 research reports, Mérat (1984) found the median depression of slaughter weight in female progeny was 0.7 percent or 10-15 g. The *dw* allele is incompletely recessive so that slaughter weight of Dw^+/dw males is not quite equal to that of normal homozygotes; in summarizing the same 14 reports, the median corresponding deviation was close to 2.8 percent of the normal genotype representing 40-60 g loss. From the same review, there was no detectable effect associated with the Dw^+/dw genotype on feed efficiency or on body fatness. A financial balance integrating all stages was found to be positive by several authors (Guillaume, 1976). Particular situations may maximize the advantages: production of broilers for early slaughter, where savings on day-old chick cost then have a larger relative influence on total cost of the broiler; broiler production in developing countries, where savings on feed costs are particularly important.

Other Genes Reducing Size

Two other genes reducing size in the fowl deserve consideration: a recessive autosomal dwarfism (*adw*), and a sex-linked factor possibly allelic with *dw* (dw^B).

Autosomal dwarfism (*adw*). This recessive gene in Leghorns reduced body weight by 30 percent, decreased hatching rate, delayed sexual maturity, caused a 10 percent reduction in egg number, but gave good viability (Cole, 1973). Its use in laying stocks for decreasing feed requirements would require introducing the allele into all parental lines of a cross. Leenstra and Pit (1984) suggested its use for reducing body size of a broiler male line mated to dwarf (*dw*) females to avoid fertility problems. In their broiler type population the *adw/adw* genotype was associated with leg abnormalities interfering with viability, but their data suggested a complete recessiveness of the *adw* gene for growth rate. The use of this gene depends on further research to determine if its influence on leg problems is permanent or caused by linkage or interaction between particular genes.

Bantam dwarfism (dw^B). Several authors (Jull and Quinn, 1931, and Maw, 1935, cited by Hutt, 1949; Godfrey, 1953; Jaap, 1969; Custodio and Jaap 1973) demonstrated the existence of a recessive sex-linked gene for small body size in bantam breeds, and located this gene near the *S* and *K* loci. Custodio and Jaap (1973) suggested this factor is an allele (dw^B) of *dw*. Telloni et al. (1973) and Shiva Prasad and Jaap (1977) found dw^B to be associated with only ten percent body size reduction and superior egg production compared to *dw* birds. Coquerelle and Mérat (1979), comparing birds carrying a similar sex-linked factor coming from the Bantam Ardennaise to normal-sized birds of similar origin, found it to be associated with a 13 percent reduction of adult body weight, mean egg weight reduced by four percent, no depression of egg number, and a feed conversion improved by six percent. Apparently in agreement with these results, Yoshida and Saito (1983) for a similar factor linked with the *K* locus observed an adult body weight reduction of 8-17 percent, a 3-4 percent smaller mean egg weight and no change in egg numbers.

Such a sex-linked gene has limited worth for improving feed efficiency by comparison with conventional selection to reduce body size. However its introduction in the male line of a cross might be relatively simple and advantageous for other reasons.

GENES AFFECTING COLOR OR MORPHOLOGY OR CAUSING ABNORMALITIES

Among genes modifying the bird's appearance, some which are sex-linked are used for sexing day-old chicks in crosses (S/s^+ and K/k^+ allelic pairs); although not having direct quantitative effects, they are of economic importance. A similar use may be found for the s^{al} (sex-linked albino) allele. Other genes with visible effect may affect heat transfer to the environment and consequently ameliorate feed efficiency. Others have direct effects on a variety of quantitative traits.

The Pea Comb Gene

Effects associated with the pea comb (*P*) gene are summarized in Table 20.5.

Several researchers (reviewed by Mérat and Bordas, 1979) comparing P/p^+ and p^+/p^+ (single comb) chickens mentioned a slight depressing effect on growth rate associated with the *P* gene; others did not. The slight depressing effect of the *P* gene on adult body weight and residual feed intake were consistent over several comparisons (Table 20.5), while egg number, egg weight, and shell strength were not affected by the P/p^+ genotype. A simple interpretation lies in the drastic reduction of comb and wattle size by the *P* gene, since these unfeathered areas may account for up to 15 percent of total heat dissipated towards the environment (Sturkie, 1965). A two percent reduction in feed intake may correspond to the lower energy than needed to maintain body temperature. This seems also in agreement with the positive correlation found by Bordas and Mérat (1981) between wattle size and residual feed consumption.

Hartmann (1972) noted a higher frequency of breast blisters in P/p^+ chicks than in single comb chicks of the same origin. The breast ridge found associated with the pea comb may be responsible for this. A slightly reduced feathering in the breast region may also contribute to this effect (Classen and Smyth, 1977).

Buckland and Hawes (1968) suggested a slight depressing effect associated with the *P* gene on male fertility. This may need additional confirmation. In pairs of P/p^+ and p^+/p^+ sibs of a medium-sized population aged 40 weeks, the former genotype was eight percent heavier (a 250 g difference) than single comb males (Mérat, 1988). Since no similar effect was noticed in females, a sex-limited hormonal effect cannot be excluded.

Only broiler strains contain the *P* gene. Two associated effects are slightly unfavorable in this context: tendency to a minor growth depression, and to more breast blisters. Conversely, the effects associated with this allele on feed efficiency and body size in layers

Table 20.5. Effects associated with the pea comb gene. (Modified from Mérat, 1970, 1988; Mérat and Bordas, 1979).

Trait	Number of populations analyzed	Deviation (percent) from the p^+/p^+ genotype
8-week weight, sexes pooled	6	-2.4 to +1.1 (median -1.5)
40-week weight, females	5	-5.4 to +0.1 (median -1.3)
Residual feed consumption, females	1	-1.8
40-week weight, males	1	+7.8

are favorable although not large, and its incorporation into laying-type stocks might avoid dubbing. In the event of selection plateaus for egg number in the future, such advantages may have increased interest.

Plumage Color Genes

Similar effects are associated with several genes suppressing or reducing eumelamins or pheomelamins in the plumage, according to reviews by Smyth (1969) and Mérat (1970). Table 20.6 summarizes growth rate comparisons between pigmented and unpigmented

Table 20.6. Effect of plumage color genes on body weight at 8 or 9 weeks of age.

	References[1]		
Comparison	Superiority of colored genotype	No significant difference	Inferiority of colored genotype
C^+/c (colored) vs. c/c (white)	Jerome and Huntsman (1959) Smyth and Fox (1961, 1963) Mérat (1965, 1967) Yamamoto et al. (1972) Mérat et al. (1979) Fox and Smyth (1982, 1985)		
C/c^+ (colored) vs. c^a/c^a (albino)	Pardue et al. (1985)		
i^+/i^+ (colored) vs. I/i^+ (white)	Jaap and Grimes (1956) Jerome et al. (1956) Collins and Hubbard (1958) Mérat (1959, 1962) Smith and Nordskog (1963) Potemkowska (1966) Yamamoto et al. (1972) Mérat et al. (1979)	Blackwood et al. (1962) Bohren and Jones (1964) Hutt (1964) Lowe et al. (1965) Fox and Smyth (1985)	Williams et al. (1959)
s^+/s^+ or $s^+/-$ (red) vs. S/s^+ or $S/-$ (non-red)	Jaap and Grimes (1956) Griesbach (1961) Knize (1967) Mérat et al. (1979)	Wessels (1963)	
E (extended black) vs. absence of *E* (non-extension)	Potemkowska (1966)		Williams et al. (1959)
Co (columbian) vs. *co* (non-columbian)	Fox and Smyth (1984)		
b^+/b^+ or $b^+/-$ (non-barred) vs. B/b^+ or $B/-$ (barred)	Jaap and Grimes (1956) Mérat (1959, 1962)	Smith and Nordskog (1963) Bohren and Jones (1964)	Williams et al. (1950) Potemkowska (1966)

[1] For references prior to 1970 refer to reviews by Smyth (1969) and Mérat (1970).

genotypes at loci *C, I, S, E* and *B*. The clearest result is a moderate inferiority consistently associated with the recessive white (*c/c*) genotype compared to the colored (C^+/c) genotype. The similarity of the results of Smyth and Fox (1961, 1963) and those of Fox and Smyth (1982, 1985) on the same population more than 20 generations later suggest a pleiotropic effect of the *c* gene. Similar results also were obtained across generations by Mérat et al. (1979). Fox and Smyth (1982) found the same growth depression associated with the *c/c* genotype when two different energy levels were fed. Pardue et al. (1985) noted a reduction of early growth for the c^a/c^a genotype (albino) at the same locus. The effects accompanying the recessive white genotype include reduced adult body weight of females; it is slightly lower than that of colored C^+/c females (Mérat, 1972; Mérat et al., 1979) at 40 weeks of age, and also at 26 weeks (Fox and Smyth, 1985), and the mean egg weight is slightly reduced for *c/c* hens.

At the *S* locus, silver birds compared to those with gold plumage had an inferior body weight in four studies and a fifth found no difference (Table 20.6). At the *I* locus, eight reports indicated a slight inferiority of dominant white chicks (I/i^+) for growth rate compared to colored chicks, five others showed no appreciable difference, and in only one was there a moderate superiority of the dominant white genotype (Table 20.6). Fox and Smyth (1985) showed that the depressed growth associated with the *c/c* genotype is expressed in presence of the *I* gene. They advise eliminating the *c* allele which may exist in broiler-type populations fixed for dominant white. For extension of black in the plumage (*E*) compared to nonextended black, and for sex-linked barring compared to nonbarring, the literature does not suggest any clear trend.

Comparisons of four loci for several traits are shown in Table 20.7. A significant depression of 8-week and 40-week weight is associated with both the recessive white and the dominant white phenotypes. The former also shows a slight reduction in egg weight. Nonsignificant effects in a similar direction concerning the colorless genotype are observed at the *S* and *Ig* loci. Residual feed intake of laying hens with recessive or dominant white plumage is slightly but significantly lower compared to their colored counterparts. Hence in these two cases noncolored hens consume less feed for the same production and body

Table 20.7. Effects on several quantitative traits associated with color suppressing genes. (Adapted from Mérat, 1968a; Mérat et al., 1979; Prod'Homme and Mérat, 1965).

Trait[1]	Deviation (percent) of unpigmented genotype from colored birds			
	c	*I*	*S*	*Ig*
8-wk wt, sexes pooled	-2.0***	-1.9**	-0.6	-0.5
Mean egg wt	-1.3***	-0.8	-0.5	-0.9
Female body wt (40 weeks)	-2.5***	-2.6***	-1.2	-1.2
Residual component of feed consumption of layers	-3.8**	-1.7*	+0.5	
Proportion of females with deteriorated plumage after 6 months of production	-25.8***	-12.9	-27.7**	-25.7

* ** *** $P<0.05$, $P<0.01$, $P<0.001$ respectively.

[1] 8 wk wt, egg wt, body wt - 23 years' data from a medium-sized strain; plumage deterioration - 4 years' data; residual feed consumption - 2 experiments for each locus.

weight. In four different years the proportion of hens with deteriorated plumage following six months in cages was lower among unpigmented genotypes at each of the four loci, significantly so for two of them (*C* and *S*). This faster deterioration of colored plumage remains unexplained. It may contribute to an increased feed intake to compensate for increased heat loss. However the birds measured for feed consumption (Table 20.7) had plumage in good condition. The records on plumage deterioration concerned other birds, so that the differences between color genotypes for feed intake require another explanation, not excluding that later an effect on plumage condition may accentuate the differences. It may be questioned whether a common factor for lower appetite in different unpigmented genotypes may cause at the same time a slightly retarded growth rate and a lower feed intake during laying. The similarity of the effect of several genes suppressing color, as well as the persistency of growth differences across generations at the *C* locus suggests a pleiotropic action associated with pigmentation.

Short down and four percent lower hatching rate was found by Hutt (1951) for embryos with extended black (*E*/*E*). The *B* and *k* genes suppress this effect. A similarly reduced hatching percentage in presence of the *E* and of the *K* alleles was obtained by Mérat (1973) and the shorter down associated with extended black was noted by Stasko et al. (1971). Bernier and Cooney (1954) showed that the black embryos with short down display symptoms of riboflavin deficiency and that extended black embryos benefit from an artificial supply of this vitamin in the air cell. Hawes and Fox (1962) partly confirmed this but found no effect of the addition of vitamin B_2 on down length. Hutt (1951) suggested that a sufficient down length may facilitate hatching in some way. This would suggest a pleiotropic nonspecific effect of the extended black gene, to be compared with the effects of other genes reducing both down length and hatching. Effects of black color on down length in the turkey (Hawes and Buss, 1963) and the domestic duck (Hawes, 1965) also suggest pleiotropy.

The knowledge of effects associated with color factors on growth, feed efficiency, or hatching rate has no practical consequence as far as the color of strains and crosses is concerned, but it may guide or it may have guided in the past the choice of specific crosses.

The Sex-Linked Albino Gene (s^{al})

The sex-linked albino gene allows day-old sexing (Hutt and Cole, 1973) so that its possible associated effects on performance traits deserve to be examined. A slightly increased late mortality among albino embryos was found by Smyth et al. (1987) but not by Champion (1958). A depression of survival rate limited to the first week of age was noted by Silversides and Crawford (1986) but not by Champion (1958) and Hutt and Cole (1973). The observed effects of s^{al} on growth and egg production traits are summarized in Table 20.8. Delayed early growth is characteristic of albino chicks; growth depression was ten percent at seven days of age but it was reduced to 1.7 percent at 56 days in a broiler-type population (Silversides and Crawford, 1986); it decreased from 10.5 percent at eight weeks to 6.1 percent at 18 weeks and 1.7 percent at 40 weeks in females in a medium-size strain (Mérat et al., 1986). The average delay in age at first egg found by Mérat et al. (1986) was three and five days in two different experiments, and for Silversides and Crawford (1986) it was five days. Average egg weight of s^{al} hens was lower than for colored hens by 1.6 and 2.1 g in two different trials (Mérat et al., 1986). For egg number, results suggest that the s^{al} allele has no consistently unfavorable effect; it may even be favorable, depending on the genetic background and/or the environment.

Table 20.8. Effects associated with the sex-linked albino gene (s^{al}) on growth and egg production traits.

Trait	Observed effect	Reference
Body weight	Delayed early growth, compensated later	Silversides and Crawford (1985, 1986)
	No significant difference for adult body wt	Silversides and Crawford (1987); Mérat et al. (1986)
Feed efficiency	Not modified	Silversides and Crawford (1986)
Egg number	No difference	Champion (1958)
	No difference (1 experiment); albinos inferior (1 experiment)	Mérat et al. (1986)
	Albinos superior	Silversides and Crawford (1987)
Age at 1st egg	Retarded by a few days	Mérat et al. (1986); Silversides and Crawford (1987)
Mean egg wt	Reduced	Mérat et al. (1986); Silversides and Crawford (1987)
Feed conversion	Not modified	Mérat et al. (1986); Silversides and Crawford (1987)

It may be questioned whether retardation of early growth and sexual maturity and inferior egg weight could be explained by impaired vision at normal light intensity. Champion (1958) and Werrett et al. (1959) suggested susceptibility of albino birds to light but others did not (Mueller and Hutt, 1941; Hutt and Cole, 1973). Smyth et al. (1987) reported a similarity of effects of the s^{al} gene and of autosomal albinism (c^a) on hatching rate as well as in causing short down and a high incidence of yolk sac protrusions. This supports Hutt's (1951) hypothesis of short down interfering with the hatching process. The parallel among several albinotic mutants suggests pleiotropic effects. The same can be said of the associations with body weight gain, age at first egg and egg weight. For these last criteria similar results were obtained with Japanese quail (Mérat et al., 1981).

From a practical point of view, the moderate but consistent depressing effect associated with the s^{al} gene on early growth does not favor its use for sexing of broiler crosses. In laying-type crosses, such use may be considered since egg number and egg mass are not consistently lowered by albinism. However further study is warranted because of conflicting results in the literature.

The Naked Neck Gene (*Na*)

Recently interest has increased concerning the naked neck gene (*Na*) because of its apparent association with heat tolerance (see reviews by Mérat, 1986; Horst and Rauen 1986; Rauen et al., 1986). This association will be described in detail below.

Growth, feed efficiency, carcass composition and meat yield. The review by Mérat (1986) includes comparisons at the *Na* locus for growth at various temperatures. In three of these the same genotypes were distributed in two environments. At 20°C or lower, the differences between genotypes were small, or naked neck birds were inferior, and feed efficiency was poor for naked neck birds. At 24-25°C, growth and feed efficiency differences between the Na/na^+ and na^+/na^+ genotypes were negligible. Near 30°C or higher, homozygous or heterozygous naked neck birds were heavier than na^+/na^+ chicks and their feed efficiency was at least equal. Figures 20.3 and 20.4 show the deviation of the *Na/Na* and Na/na^+ genotypes in percent from na^+/na^+ homozygotes according to ambient temperature after the age of four weeks for body weight and feed conversion.

The yield of carcasses is improved in two ways by the *Na* gene regardless of ambient temperature (Mérat, 1986). Reduction of plumage (around 30 percent for Na/na^+ and 40 percent for *Na/Na*) causes gains of 1.5-2.0 percent and 2.5-3.0 percent in slaughter yield of the two genotypes respectively. There is also an increase in meat yield of dressed carcasses. The advantage of Na/na^+ over na^+/na^+ in this respect was 1.8-7.1 percent in males and 0.8-5.1 percent in females according to the population studied. This resulted from a higher proportion of muscle in naked neck chicks in the pectoral region. There was no difference between Na/na^+ and wild-type chicks for percent abdominal fat, but there was a lower percentage of intermuscular and subcutaneous fat in the naked neck birds. This is in agreement with another study which demonstrated lower body fat content in naked neck broilers.

Egg production and efficiency traits. The results reviewed by Mérat (1986), Horst and Rauen (1986), and Rauen et al. (1986) showed a different response of the naked neck (*Na/Na or* Na/na^+) and normal genotypes to high environmental temperature. Table 20.9 illustrates the mean deviations of naked neck types from the na^+/na^+ genotype at two temperatures in four experiments.

Egg numbers at moderate temperature are not significantly affected by the naked neck gene. At high temperature naked neck hens mainly in Experiment 4 seemed to have a better laying rate. In Experiments 1 and 2 at 20°C adult body weight was lower in naked neck hens, especially in homozygotes, than in hens with complete plumage, the tendency being slightly reversed above 30°C. Consequently, although the ratio of egg weight to body weight is increased by the *Na* gene at any temperature, the increase of mean egg weight in *Na/Na* and Na/na^+ genotypes compared with na^+/na^+ is lower at moderate temperature than

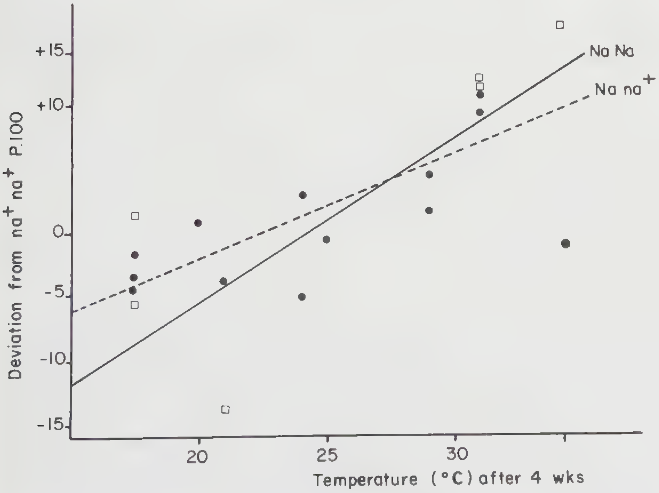

Figure 20.3. Body weight at 8 or 10 wks of *Na/Na* (□) or Na/na^+ (•) chicks according to temperature. (Adapted from five research reports for *Na/Na* and ten for Na/na^+).

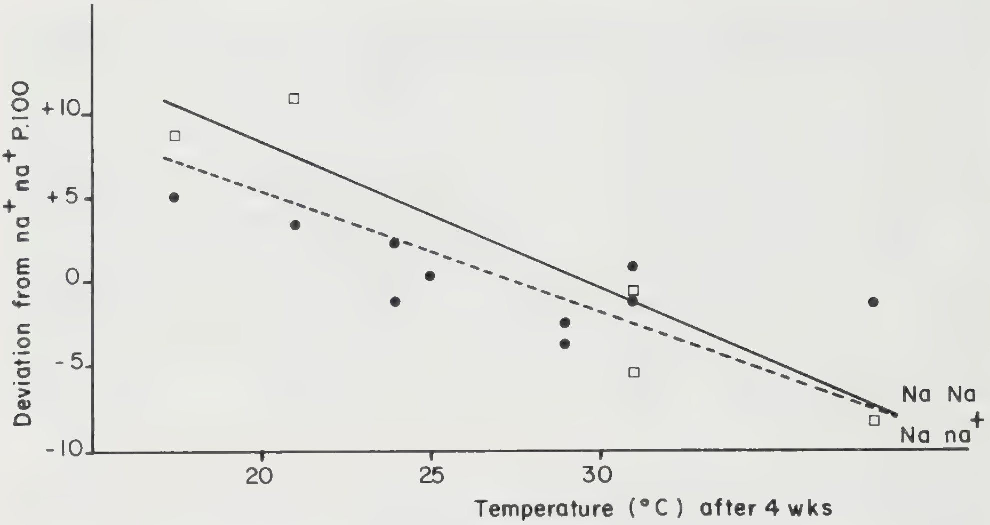

Figure 20.4. Feed conversion to 8 or 10 wks of *Na/Na* (□) or *Na/na*$^+$ (•) chicks according to temperature. (Adapted from five research reports for *Na/Na* and ten for *Na/na*$^+$).

at high temperature where it reaches 3-4 g for the naked neck homozygote. The trends for egg mass are the same. Concerning feed efficiency, for naked neck females above 30°C the residual component of feed consumption (at equivalent body weight and production) is not higher than that of *na*$^+$/*na*$^+$ hens, while in the temperate environment this variable is increased because of greater heat dissipation. The unfavorable effect of higher residual feed intake of naked neck layers is compensated at moderate temperature by lower body weight for *Na/na*$^+$ but not for *Na/Na*. Above 30°C feed efficiency is always superior for naked neck females.

The percentage of cracked eggs was not significantly different for genotypes at the *Na* locus in the above experiments. However, the mean values suggest a slight disadvantage for the *Na/Na* genotype at the lower temperature and a slight advantage at the warmer temperature.

At a constant temperature of 25°C no significant effect is associated with the *Na/Na* and *Na/na*$^+$ genotypes for body weight, egg number and feed conversion (Table 20.10). The mean egg weight and egg mass are increased as at the other temperatures. Fraga and Lam (1987) with similar mean ambient temperatures found the same, and in addition they found better egg shell strength for the *Na/na*$^+$ genotype.

An interaction appears between the *Na* and *dw* genes. In Experiment 2 of Table 20.9 concerning a dwarf line and in previous results (Mérat, 1986), the efficiency at moderate temperatures is not worsened even for the *Na/Na* genotype in comparison with *na*$^+$/*na*$^+$. The opposite was found in Experiment 1 using *Dw*$^+$ hens, since the increase of the residual fraction of feed consumption associated with the *Na/na*$^+$ and *Na/Na* genotypes was lower when the sex-linked dwarf gene *dw* was present rather than *Dw*$^+$. On the other hand, in a dwarf line at 23-24°C (Mérat, unpublished), the *Na/na*$^+$ genotype had a 5.5 percent higher egg number to 39 weeks of age than the normally feathered genotype. This and data in Table 20.10 suggest that in a dwarf background and in a moderately warm environment the *Na/na*$^+$ genotype has an advantage for egg production.

Table 20.9. Comparison between genotypes at the *Na* locus for egg production and efficiency traits at two temperatures in four experiments, percent deviation from na^{+}/na^{+}.

	Experiment[1]					
	1		2		3	4
	Na/Na	Na/na^{+}	Na/Na	Na/na^{+}	Na/na^{+}	Na/na^{+}
	Moderate temperatures					
Trait	15-20°C		15-20°C		18-21°C	18-21°C
Mature body wt	-10.5	-6.1	-13.9	-5.9		
Egg number	-3.8	-1.1	-3.3	+2.0	-1.8	-1.7
Mean egg wt	+4.4	+0.4	+1.4	+2.3	+1.0	+1.3
Egg wt/body wt	+16.9	+6.7	+17.7	+8.7		
Egg mass	+1.7	+4.2	+1.0	+6.5	+15.8	-0.4
Residual feed intake (percent of total feed of na^{+}/na^{+})	+17.9	+4.6	+3.9	+1.3		
Feed efficiency (g eggs/g feed)	-8.3	+8.5	+6.8	+10.3	-3.3	-3.9
	High temperatures					
	31°C		31°C		32°C	32°C
Mature body wt	+5.5	+3.4	+0.3	+0.3		
Egg number	+4.7	+2.2	+2.4	-0.6	+4.8	+16.2
Mean egg wt	+9.2	+6.2	+7.1	+2.3	+3.0	+6.7
Egg wt/body wt	+4.2	+3.1	+6.5	+1.7		
Egg mass	+10.5	+4.9	+22.9	+11.6	+8.3	+23.2
Residual feed intake	-0.8	-1.6	+0.0	-0.5		
Feed efficiency	+0.3	-0.8	+13.4	+7.4	+1.3	+9.9

[1] Experiment 1 - Dw^{+} medium-sized type (Bordas et al., 1980).
Experiment 2 - *dw* medium-sized type (Bordas and Mérat, 1984).
Experiment 3 & 4 - Dw^{+} medium-sized and light types (Rauen et al., 1986).

Rauen et al. (1986) observed that the advantage of the *Na* gene at high temperature for egg production mainly involves persistency of laying and is more marked in medium-sized than in light strains.

Reproduction and viability. Smith and Lee (1977) observed no difference between Na/na^{+} and na^{+}/na^{+} males for fertility and semen traits. Hammade et al. (1987) compared males kept in cages at 18°C and 30°C; they obtained at successive ages a larger semen volume and number of spermatozoa per ejaculate for Na/Na than for na^{+}/na^{+} birds, with intermediate values for heterozygotes and without genotype x temperature interaction. No significant differences between genotypes appeared for fertility.

An increase of embryonic mortality (up to ten percent in pure strains) was found associated with Na/Na and Na/na^{+} genotypes by Crawford (1977, 1978), Horst (1982),

Table 20.10. Comparison between genotypes at the *Na* locus for egg production and efficiency traits at 25°C, percent deviation from mean of na^+/na^+. (Adapted from Mérat, 1986).

	Experiment[1]			
	1		2	
Trait	*Na/Na*	Na/na^+	*Na/Na*	Na/na^+
Mature body wt	-3.3	+1.2	+0.2	+0.6
Egg number	-2.4	+2.6	+2.9	+5.3
Mean egg wt	+1.8	+2.7	+2.5	+1.4
Egg mass	+0.3	+6.9	+8.6	+6.9
Feed efficiency (g eggs/g feed)	-1.7	+1.4	-1.2	-1.5

[1] Experiments 1 and 2 - all birds were *dw* medium-sized type.

Mérat (1986), and Rauen (1985). The losses occurred mostly during the last stages before hatching.

Postembryonic chick mortality has not been found different for naked neck or fully feathered chicks, except when exposed to heat stress above 40°C, where Smith and Lee (1977) noticed a survival rate of 51.4 percent for Na/na^+ and 38.8 percent for na^+/na^+ birds. In a similar experiment on two-day old chicks (Mérat, 1986) the survival percentages of the same genotypes were 59.3 and 33.3 percent respectively.

Mortality of adults does not differ between the naked neck and normally feathered types around 20°C. At 30°C or more, mortality was lower for heterozygous naked neck layers than for comparable na^+/na^+ hens (Rauen et al., 1986). A difference in the same direction exists at 25°C between dwarf lines differing in genotype at the *Na* locus (Table 20.11). Results from Barrio et al. (1987) indicate less frequent cannibalism in presence of the *Na* gene, which may also have a relation to survival rate.

Interpretation of associated effects. Reduction of the pterylae by the *Na* gene is unexplained but many of the secondary effects of *Na* are interpreted through this reduction. A consequence of it is increased slaughter yield after plucking. Other consequences arise from heat loss through an increased surface deprived of plumage cover. Feed consumption is raised at any temperature, and this may account for the increased egg weight because of greater ingestion of nutrients, especially proteins.

At moderate or low temperatures this overconsumption associated with a higher energy requirement leads to poor feed efficiency, while at elevated ambient temperatures a faster heat dissipation permits sufficient feed intake for a high growth rate or laying intensity, it may maintain egg shell quality by preserving the acid-base balance, and it may lower mortality from heat stress.

Some effects associated with the *Na* gene are puzzling, such as depression of body weight at low temperatures. An insufficient compensation for energy needs is suggested by the slight reduction of body temperature (about 0.2°C) in naked neck birds (Mérat, 1986). The increased percentage of muscle is not clearly explained; it may be questioned whether

Table 20.11. Adult mortality - comparison of *Na* locus genotypes at different ambient temperatures.

		Genotype					
		Na/Na		*Na/na*$^+$		*na*$^+$*/na*$^+$	
Reference	Temperature	n	Mortality, %	n	Mortality, %	n	Mortality, %
Rauen (1985)							
Exp. 1	control			220	5.9	259	5.8
	warm (32°C)			215	14.4	259	24.0
Exp. 2	control			233	2.1	248	3.6
	warm (32°C)			230	8.3	251	20.9
Boichard and Mérat (unpublished)							
	moderate (25°C)	515	5.4	563	8.7	564	10.5

more protein material is left available for muscle development through lower requirements for plumage growth.

The high late embryonic mortality associated with the *Na* gene has not been fully explained. According to Bernon and Crawford (1988, personal communication), much of the reduced hatchability seems to be due to malposition of embryos, perhaps due to few feathers on the neck allowing the head to assume faulty positions. The lower proportion of subcutaneous and intermuscular fat (see Mérat, 1986) in naked neck chicks may result from the utilization of a larger fraction of energy for thermoregulation. Concerning naked neck adult males, the slight depression of their body temperature may contribute to their higher semen production, since it is known that high temperatures have a depressing influence (de Reviers, 1982). Finally, as concerns egg weight it is interesting that two other genes suppressing or reducing the plumage, *sc* (Abbott, 1958; Abbott and Asmundson, 1962) and K^n (Somes, 1975), are associated with a mean egg weight higher by several grams.

The hypotheses discussed above are summarized in Figure 20.5.

Potential usefulness of the naked neck gene in poultry production. The most likely use of the naked neck gene is at high mean ambient temperatures, 25°C and above, where its advantages concern higher growth rate, slaughter yield, meat yield, and resistance to acute heat stress. In addition, faster plucking leaving fewer pinfeathers may be expected, along with a slight positive maternal effect on early growth caused by higher mean egg weight if the *Na* allele is incorporated into the dam line. Further work will have to establish whether the advantages observed for the *Na/Na* or *Na/na*$^+$ genotypes at constant high temperatures also occur in fluctuating temperature environments and at various relative humidities. The depression of hatching rate for naked neck birds would be the only unfavorable effect associated with the naked neck character in a hot environment. The incubation loss associated with the naked neck gene might be lower in F_1 crosses than in a pure strain according to field observations, but its economic impact may not be compensated by gains in other traits (Mérat, 1986).

For egg production at high temperature, most important are lower mortality for naked neck hens than for *na*$^+$*/na*$^+$ ones, higher mean egg weight, and possibly better shell strength and laying persistency. These benefits should also apply to female broiler breeders.

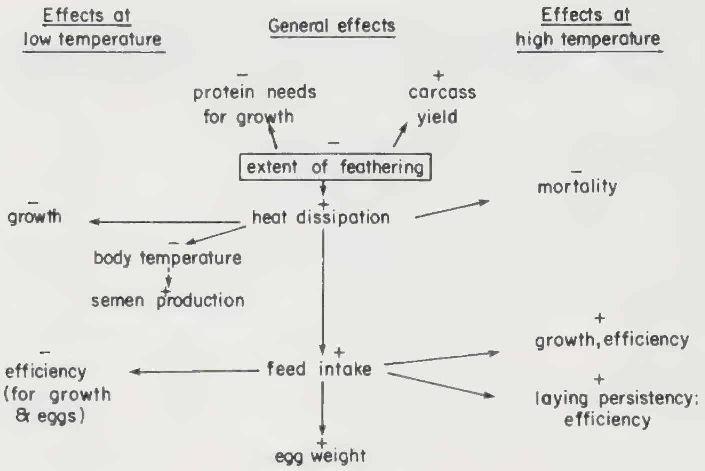

Figure 20.5. Supposed causal links between effects (+ or -) associated with the *Na* gene.

While at 15 to 20°C naked neck chickens are clearly inferior for broiler production, a comparison at 25°C in a cross between Cornish males and Na/na^+ females showed the same eight-week body weight and the same feed conversion for Na/na^+ and na^+/na^+ birds (Mérat, 1986). In such conditions the advantage of Na/na^+ birds for slaughter yield and meat yield is to be compared with the cost of additional heating and with the loss in hatching rate associated with the *Na* gene. As suggested (Mérat et al., 1981), this balance could be more favorable for naked neck chicks if slaughter age is early so that the duration of additional heating is minimized.

For egg production, on a dwarf (*dw*) background a limited increase in feed intake and better laying rate associated with the *Na* gene make the balance positive for the Na/na^+ genotype. Further work however should estimate more accurately the overall effect on feed efficiency in a dwarf stock and on shell strength for naked neck birds under temperate conditions.

In conclusion, more research seems justified to establish in greater detail the environment in which the naked neck gene may be beneficial for broiler or egg production, and to determine the economic interest of incorporating naked neck into commercial parental lines. More research is also needed on the suggestions that for broiler production a naked neck cross devised for hot climates might be used in temperate conditions with early slaughter and heating to around 25°C, and that for egg production a dwarf naked neck layer for predominantly hot areas might combine the advantages of both genes.

Quantitative Effects Associated With Other Marker Genes

Frizzled plumage (*F*) and thermal insulation. Among other genes likely to diminish the insulating power of the plumage is the frizzle (*F*) gene. Very few data are available on its possible effects on production traits. Hutt (1949) mentioned that hatchability is less good for frizzle embryos, and suggested a retarded sexual maturity and possibly a higher mortality than for the normal genotype. Benedict et al. (1932) found a considerable increase in energy metabolism for frizzled birds, suggesting that they will respond differently from normal birds to hot temperatures; the same holds for the observation by Hutt (1949) of less evaporative loss from frizzled birds. However experimental data are still scanty for determining if frizzle plumage may have an advantage in a hot climatic environment. According to Horst (1987) and Haaren-Kiso et al. (1988), under hot conditions the *F* gene increases egg number and egg mass and reduces mortality, and the double heterozygote condition for *Na* and *F* loci seems to be favorable for egg laying traits.

Rose comb and male fertility. A large single gene effect is the poor fertility of males homozygous for the rose comb gene (Cochez, 1951; Crawford and Merritt, 1963). Mating frequency is lower (Fox et al., 1964; Crawford and Smyth, 1965; Cook and Siegel, 1973) and duration of fertility after a single insemination is shorter (Crawford and Smyth, 1964a) for *R*/*R* males. The motility of their spermatozoa after storage is inferior (Petitjean and Cochez, 1966) and certain enzymatic activities differ (Buckland et al., 1968; Petitjean and Servouse, 1981) from R/r^+ and r^+/r^+ males.

The origin of the subfertility of *R*/*R* males remains unknown but Crawford and Smyth (1964b) found evidence that it was not due to the genotype of the sperm cells but instead it was due to deleterious effects of the *R*/*R* host body on the spermatozoa. The *He* locus which modifies the appearance of rose comb may also be associated with a difference in male fertility (Cavalie and Mérat, 1965).

Sex-linked slow feathering (*K*), and linkage with *ev-21*. Results reviewed by Mérat (1970) concerning the growth rate of rapid (k^+) and slow (*K*) feathering chicks showed either no difference, or a slight superiority for rapid-feathering chicks. Verma and Prasad (1981) obtained a more rapid growth for late-feathering chicks and according to Dunnington and Siegel (1986) k^+ females were slightly heavier than *K* females in a broiler strain but not in two divergent body weight lines. Dunnington et al. (1987) observed heavier slow-feathering males than rapid-feathering ones when fed one day out of two, but not with *ad libitum* feeding. McDonald (1958) found a difference in the conversion of methionine into cystine between females in reciprocal crosses of a fast-feathering with a slow-feathering strain, suggesting an association with the K/k^+ allelic pair, but this was not directly verified. For daughters of k^+ and *K* dams mated to the same males an advantage for eight-week weight was associated with the k^+ maternal genotype. Similar results from several strains suggested a pleiotropic effect (Mérat, 1967).

For egg production criteria, most reports show no association with the *K* or k^+ alleles, except that Lowe and Garwood (1976) and Verma and Singh (1983) found a later age at first egg and slightly lower hen-day production for late-feathering hens. Lowe and Garwood (1981) found no difference for the same traits but hen-housed production was lower for late-feathering females. In Dunnington and Siegel's (1986) data no difference associated with feathering alleles appears for laying traits. Hence if a depressive effect of the *K* allele on egg production is noticed in some cases it is not general.

However, in recent years utilization of the *K* gene for day-old sexing of egg production crosses has revealed lowered laying performance and higher mortality following the use of this gene in a maternal line. In the data of Mérat (1967) there was a delay in sexual maturity with progeny of *K* dams. Lowe and Garwood (1981) found a negative maternal effect of the *K* allele on 40-week body weight and viability. More specifically Harris et al. (1984) showed a higher susceptibility of female progeny from *K* dams to leukosis but the genotype of the progeny also influenced susceptibility, *K* daughters being more susceptible than k^+ progeny (although it was not the case in one later trial reported by Bacon et al., 1986). Havenstein et al. (1987) also observed lower egg production in the progeny when the dam was of the *K* genotype.

The work of Bacon et al. (1985, 1986) in Leghorn stocks, showing linkage of the *K* allele with an endogenous proviral gene (*ev-21*) which may interfere with immune response of the progeny against leukosis viruses transmitted through the egg, suggests an explanation for lower egg production and/or survival rate of this progeny. There is no difference associated with the K/k^+ alleles as regards stress response (Dunnington et al., 1987), or immune response to other antigens (Dunnington and Siegel, 1986), or Marek's disease virus (Somes and Jakowski, 1974). Dunnington et al. (1986) suggested a slight advantage of k^+ over *K* birds for resistance to *E. coli.*

Other marker genes. Associated effects of several other major or marker genes are summarized in Table 20.12.

Lethal genes. Lethal genes have economic implications since in the homozygous state they interfere with hatching or posthatching survival. However each lethal gene is often limited to a single population, and generally the eradication of lethal genes is realized by conventional selection on reproduction or overall survival rate. A problem may be posed if the heterozygous genotype for a lethal gene differs from the normal homozygote for performance traits. A case such as this was reported by Bernier (1951) concerning the mutation micromelia VII (*mm-VII*) in a White Leghorn strain; egg production was observed to be ten percent higher for hens heterozygous for the *mm-VII* gene compared to normal homozygotes.

Table 20.12. Quantitative effects associated with other major or marker genes.

Gene	Associated effect	Reference
O (blue eggshell)	Lower percent cracked eggs, shell thickness not affected	Silber and Mérat (1974)
	No difference in shell quality but lower percent production	Sadjadi et al. (1983b)
Mb (muffs and beard)	Thinner egg shells	Crawford (1983)
R (rose comb)	Lower 8-week weight for progeny of rose comb dams	Mérat (1967, 1968b)
W (white skin)	Lower 8-week weight for progeny of *W/W* dams	
id^c (green shanks)	Increased mortality and tumors	McGibbon (1975, 1979)
D (duplex comb)	Resistance to Marek's disease infection	Somes and Jakowski (1974)
Bl (blue plumage)	Lower overall adult hen mortality for Bl/bl^+ than bl^+/bl^+ females	Mérat (1973)

BIOCHEMICAL POLYMORPHISMS

The literature on associations between biochemical (mainly electrophoretic) polymorphisms with a Mendelian basis and quantitative traits is abundant. But it is often difficult to draw firm conclusions from results that vary in statistical significance and that sometimes are conflicting. It is true that the likelihood of finding such associations is low. In a review, Mina (1978) remarked that among data on five egg protein and serum protein loci and 23 traits the total number of significant results did not exceed that to be expected from random variation alone. However, this does not preclude that a few of the observed differences may be too highly significant to be easily accounted for in any other way. Furthermore some general trends, even of limited scope, may be looked for. Without being exhaustive, comparisons have been assembled concerning egg white protein loci (Table 20.13), blood protein loci (Table 20.14), and isoenzymes (Table 20.15).

For egg white proteins and enzymes, genotypic differences for a given trait are often nonsignificant or variable with complex interactions. In addition, for reproductive traits direct and maternal effects are not always clearly separated. However a few general trends are suggested and the following tentative conclusions are proposed:

1. For the ovalbumin locus, differences between genotypes for body weights, production traits, and reproduction traits are only occasionally significant, but when such is the case the suggested advantage is for the *AA* homozygote.
2. At the *Tf* locus the situation seems analogous for production traits and body weight, with a suggested advantage for the *BB* homozygote when a significant difference is noted. For hatchability, results are conflicting, with the *BB* or the *AB* genotype being occasionally superior.
3. The G_3 locus shows a variable situation for production and reproduction traits, at times with suggested overdominance, or with an advantage for the *AA* homozygote, which may imply interactions with genetic or environmental background. The situation for the G_2 locus with fewer reported results seems similar. However, there are several suggestions of overdominance at the G_3 locus with higher resistance of the *AB* genotype against *Salmonella* or coccidiosis infection. It may be questioned if the occasional heterozygote advantage suggested for egg production traits may represent situations of latent infection, or if differences in disease resistance may reflect general nonspecific overdominance.
4. The hemoglobin mutant extensively studied by Washburn et al. (1971) and Washburn (1975) shows a higher resistance to Marek's disease but lower egg production, and the seemingly similar mutant studied by Shabalina (1976) is associated with lower hatchability.
5. The *F* and *S* alleles for alkaline phosphatase show several conflicting results, some suggesting superiority of the fast phenotype for egg production traits, others suggesting the reverse. Linkage may be involved. A search for gene x genome and/or gene x environment interactions might be justified.
6. The cholinesterase locus seems to be associated with differences in resistance to pathogens such as *Salmonella* spp. and Marek's disease virus.

It is tempting to make a parallel between suggested trends in relation to some production traits and the general picture for allelic frequencies at polymorphic loci. It has long been observed (Manwell and Baker, 1970 and others) that at the *Ov* and *Tf* loci one allele is generally predominant, which may be in agreement with occasional associated advantage. Conversely at the G_3, alkaline phosphatase, and esterase loci a polymorphism is

Table 20.13. Egg white protein genotypes and economic traits.

Trait	Reference	Nature of result
OVALBUMIN (*Ov*) LOCUS		
Quantitative Traits		
Adult body weight	Buvanendran (1967b)	Additive effect, *A* allele superior
Egg production traits	Durand and Mérat (1988)	Progeny of *AA* dams heavier
	Mina (1976)	Interactions between loci
Hatchability	Stratil (1967)	*AA* superior to *AB* and *BB*
	Kushner et al. (1974)	*AA* superior (in 1 line) to *AB* and *BB*
	Moiseeva et al. (1977)	*AA* superior to *AB* and *BB*
G_2 AND G_3 LOCI		
Quantitative Traits		
Egg number	Buvanendran (1967b)	G_3 : overdominance in 2 populations
	Titok (1970)	G_3 : *AA* superior to *AB* G_2 : *DD* superior to *DC*
Gene frequency	Zui and Tyshkovets (1971)	G_3 : *A* more frequent in laying stock, less in broiler stocks
Age at first egg	Ermenkova (1978)	G_3 and G_2 : overdominance
Egg weight	Obeidah et al. (1977)	G_2 : *AB* superior to *BB*
Growth rate	Kuryl (1979)	G_3 : no differences
	Csuka and Baumgartner (1978)	G_3 : no differences
Fertility and Hatchability		
Hatchability	Buvanendran (1967a)	G_3 : different effects in 2 lines and 2 generations
	Stratil (1967)	G_3 and G_2 : *AB* superior
Fertility, hatchability	Schmid & Thein (1968, 1969)	*A* allele superior in some lines
	Juneja and Chaudhary (1972)	G_3 and G_2 : *AB* inferior
	Durand and Mérat (1982)	G_3 : *AA* or *AB* dams superior to *BB*
	Morton et al. (1965)	Complex interactions
	Pavel and Peterson (1969)	G_3 : *AB* dams more fertile G_2 : inferior hatchability for *AB* dams
	Kushner et al. (1974)	G_3 : *AB* superior (heavy strain) G_2 : *BB* superior (light strain)
	Mulsow and Neumann (1979)	G_3 : *AB* dams superior
	Moiseeva et al. (1977)	G_3 : *AA* superior
Fertility	Titok (1970)	G_3 : *AA* superior G_2 : *DD* superior
Disease Resistance and Mortality		
Coccidiosis	Petrovsky et al. (1968)	G_3 : different frequencies in resistant/susceptible lines
Salmonella pullorum,	Meleko (1977)	G_3 : progeny of *AB* dams more resistant
Salmonella gallinarum	Iotova (1983)	G_3 : *AB* more resistant in 3 breeds

often or generally found, reflecting possible heterozygote advantage.

From a practical viewpoint, even if associated effects on production or reproduction traits exist for egg white protein or enzyme loci, they represent only a limited fraction of the total variance of quantitative traits (Sheldon, 1980) and their use in the improvement of such traits is not likely. More interesting are the suggested relations of G_3 and esterase loci with disease resistance since direct selection in this area is difficult. (In the pigeon

Table 20.14. Blood protein genotypes and economic traits.

Protein and trait	Reference	Nature of the effect
HEMOGLOBIN		
Egg production	Lowe and Washburn (1971)	Mutant homozygote : fewer eggs
Resistance to Marek's disease	Washburn et al. (1971)	Mutant more resistant
Resistance to coccidiosis	Washburn (1975)	No difference
Hatchability	Shabalina (1976)	Mutant : lower hatchability
TRANSFERRIN		
Egg production	Buvanendran (1967b)	*BB* superior to *AB*, *AA*
	Jain et al. (1980)	*BB* superior to *AB* and *AA*
Adult body weight	Rashid (1982)	*BB* superior to *AB* and *AA*
	Durand and Mérat (1988)	Progeny of *BB* dams heavier than for *AB* dams
Fertility and hatchability	Gilmour and Morton (1971)	Maternal effect : interaction with other loci
	Titok (1971)	*AA* x *AA* matings give lower fertility, *AB* embryos have better hatchability
Hatchability, early survival	Kromin and Kovalenko (1971)	*AA* dams give lower hatchability and early survival than *AB* or *BB*
	Vichinski (1969)	*AB* x *AB* matings give better hatchability; *AB* embryos have better hatchability but poorer early survival than *AA*, *BB*

Frelinger (1972) showed more antibacterial effect of egg albumen from females heterozygous at the transferrin locus; in the fowl there are no similar reports). A deeper insight would be needed into the possible mechanisms of associated resistance to determine if it might be used in breeding programs for resistance to pathogens. It is too early to say if more general polymorphisms like that for alkaline phosphatase, which concerns many steps in metabolism (Bell, 1972), may play a role in adaptation to specific nutritional or environmental conditions.

BLOOD GROUPS

Major histocompatibility complex (*B* locus). There is a very large body of literature on the major histocompatibility complex (*B* locus). It is reviewed in Chapter 22, and aspects concerning its role in genetic resistance to disease are reviewed in Chapter 33. Comments here will be restricted to relationship of the *B* locus to economic performance.

The relationship of *B* blood-types with production traits has been under investigation for a long time. Pioneer work was done in particular by Briles and associates, as summarized in Briles (1960), Gilmour (1960), and Nordskog (1964). These and more recent studies are considered here only in a very general way.

For various traits and in diverse populations, a heterozygote advantage was sometimes found (Briles, 1960, 1964; Gilmour, 1960; and others). Most often this was due to inferiority of homozygous genotypes. Comparisons were mainly done within inbred lines. This heterozygote superiority may be at least part of the explanation for persistence of polymorphisms at the *B* locus even in highly inbred lines (Briles et al., 1957; Gilmour,

Table 20.15. Isoenzymes and economic traits.

Enzyme and trait	Reference	Nature of the effect
ALKALINE PHOSPHATASE		
Egg production traits	Rao et al. (1980)	No difference
6-week weight	Wilcox (1966)	Fast phenotype slightly superior
Albumen quality, blood spots in eggs	Engh (1966)	Fast : lower quality, more blood spots
Egg production	Chaudhary (1972)	Fast phenotype superior
	Gasparska et al. (1980)	Slow allele more frequent among poor layers; no significant differences between types
	Patil et al. (1981)	No difference in frequencies according to laying rate
Egg production, semen production	Pingel and Ranjan (1975), Ranjan et al. (1974)	Slow allele superior
Egg weight and production	Jain et al. (1976)	Fast phenotype superior
Growth, egg production	Shukla et al. (1982)	No difference
Early egg production	Amin et al. (1980)	No difference
Marek's disease resistance	Grunder et al. (1969)	No difference
	Okada et al. (1977)	Change in frequency in 2 lines out of 7 selected for resistance
CHOLINESTERASE		
Growth rate	Grunder and Merritt (1977)	Different frequencies in growth selected and control lines
DISEASE RESISTANCE		
Salmonella pullorum, Salmonella gallinarum	Shabalina and Iotova (1977)	Heterozygotes more resistant
Marek's disease	Iotsov et al. (1982)	Heterozygotes more resistant
LIVER ESTERASE	Maeda et al. (1978)	Differences in fertility, age at first egg
ORNITHIN TRANSCARBAMYLASE		
Egg production	Tsuji et al. (1978), Tsuji and Fukushima (1980)	Effect on egg mass

1959, 1960).

Since crosses are used for production, the above observation does not have direct practical bearing. Of more direct application is the observation in outbred populations or crosses that specific heterozygotes have better performance than others (Briles, 1960 for hatchability; Allen and Gilmour, 1962 for livability).

Interactions have been found for performance traits between *B* haplotypes and year (Briles, 1964; Hardin, 1971), hatch (Briles, 1964), and genes at other loci (Morton et al., 1965). Differences in livability have sometimes been reversed, depending on stage of the life cycle (Briles et al., 1957). Such lability renders difficult the interpretation and use of effects associated with *B* genotypes.

According to Gavora and Spencer (1979) there is a negative genetic correlation between resistance to Marek's disease and body weight and egg weight, and a positive genetic correlation with egg production. If this applies generally to *B* haplotypes it may be a useful guide to their choice in a breeding program.

It has been argued that observed relationships between *B* genotypes and production traits may reflect their effect on mortality or morbidity (see Briles, 1964). Bacon (1987) remarked that in recent years the use of 'clean' animals, especially concerning Marek's disease, renders it more likely that some differences in production traits can be independent of infection. In addition it should be noted that the B^{21} allele, known for its association with resistance to Marek's disease, is not appreciably superior for egg production traits according to McDermid (1964) and Nordskog et al. (1973).

Maternal effects for livability and hatchability were sometimes found in association with *B* genotypes (see Briles, 1960). The observation by Bacon (1987) of differences between maternal *B* genotypes for shedding of leukosis viruses in the eggs should be taken into account in this respect.

Other genes identified by serological methods. Blood groups other than the *B* complex have received little investigation concerning their possible relationship with production performance or disease resistance. Shultz and Briles (1953) suggested that selection for egg number in inbred lines was favorable to heterozygotes at the *A* locus, but direct comparisons between genotypes failed to show significant differences. Gilmour (1959) for the same locus found a suggestion of negative heterosis. Osterlee et al. (1971) found a relationship between *A* alleles and fertility. De Silva (1965) obtained low fertility for matings between *L* locus homozygotes in inbred strains.

The *Hi* gene concerns an agglutination reaction with seed extracts, especially *Lens culinaris*. Hens with red blood cells that are agglutinated have the dominant allele (*Hi*) and hens without agglutination are of the *hi*/*hi* genotype. The reaction takes place only when there is a sufficient estrogen level in the blood (Scheinberg and Reckel, 1962). In a brown egg population Durand and Mérat (1971) found a significant superiority of phenotypically positive females for hen-day production during the first half of the laying year. This advantage was seen in the heterozygous genotype compared to both homozygotes (Durand and Mérat, 1973), with a reduction in production pauses (Durand and Mérat, 1976). These results were consistent over several generations. However no significant differences have appeared in a Leghorn strain (Durand and Mérat, unpublished). However, Petrovsky et al. (1968) obtained superior egg production after ten months laying for Leghorn hens not exhibiting the *Hi* phenotype. These apparently conflicting results may suggest linkage between the *Hi* locus and genes affecting egg production, and/or a differential effect according to the stage of laying.

Little information is available concerning antigens not associated with red blood cells and relationship with quantitative traits. For γ-globulin allotypes the finding by David et al. (1966) of large differences in allele frequencies between lines selected for high or low egg production or high or low egg weight suggest that the alleles may not be neutral with respect to the selected traits.

ASSOCIATED EFFECTS IN OTHER SPECIES

Japanese Quail

A few mutations in Japanese quail have been investigated for associated quantitative effects. Refer to Chapter 13 for detailed description of these variants.

Imperfect albinism (*al*). In a segregating population, a comparison of albino vs.

wild-type quail repeated over two generations (Mérat et al., 1981) revealed a number of effects associated with the albino mutation. There was more than 30 percent posthatching mortality, mainly during the first week of life, but no influence on mortality after five weeks of age. Early growth was delayed by 13.6 percent at 23 days of age but the difference was reduced to 2.7 percent at 93 days of age. Egg number and age at first egg were not appreciably affected, but mean egg weight was slightly lowered and yolk percentage was higher for albino females. Feed consumption of albino layers was six percent lower than that of wild-type females and residual feed intake (for same body weight and egg mass) was four percent lower too. The albino quail were less active than colored ones. These results are very similar to those found with the homologous mutant in domestic fowl.

White egg-shell (*we*). Comparisons in a segregating population between females homozygous for the white egg-shell mutant and heterozygous females with pigmented shells showed no significant differences for body weight at different ages, age at sexual maturity, egg production to eight months of age, and egg weight (Mérat, unpublished). The only significant differences concerned egg shell thickness, which was about ten percent lower for white than for pigmented shells, and egg shell weight. Percent cracked eggs was slightly higher for eggs with white shells. There was no difference in water loss during incubation. A comparison between reciprocal crosses showed that when the dam was homozygous for white shell, the hatching percentage was 65.8 vs 81.5 for the reciprocal. Progeny of white shell dams had lower eight-week body weight and delayed age at first egg, suggesting a maternal effect associated with the *we* gene.

Hemoglobin ($Hb\text{-}1^A$, $Hb\text{-}1^B$). The electrophoretic variants for hemoglobin of the Japanese quail, A and B, were compared by Maeda et al. (1977a) with the conclusion that the *BB* genotype was inferior to *AB* for growth rate to six weeks of age, posthatch mortality, egg production, and fertility. *B* had a low frequency in diverse populations (Maeda et al., 1975). Dimri et al. (1981) found the *B* allele to be associated with lower hatchability.

Rough-textured feathers (*rt*). A maternal effect was found associated with this recessive mutant by Roberts and Fulton (1979), the viability of embryos from *rt/rt* females being reduced.

Acid phosphatase-2 ($Acp\text{-}2^A$, $Acp\text{-}2^B$). The body weight of *AB* birds was found lighter than that of *AA* or *BB* birds in both sexes (Maeda et al., 1977b).

Turkey

Only a few mutants in turkeys appear to have associated quantitative effects. Refer to Chapter 12 for a detailed description of these mutants.

Association of black color with clubbed down in turkeys was reported by Hawes and Buss (1963). The relationship is similar to that described earlier for extension of black in domestic fowl. Hawes (1965) found the same phenomenon expressed in domestic ducks. Asmundson (1950) reported that the brown gene (*e*) had no apparent detrimental effect on survival or growth. Buss (1960) studied turkeys segregating at three plumage color loci (spotted, recessive white, and grey). For each locus he found that heterozygotes were heavier than recessive homozygotes.

Buss (1974, personal communication) also found differences in 24-week body weight associated with six color genes in the turkey. Nestor and Renner (1979) found that the red mutant was associated with lower body weight and egg production, the spotted mutant was associated with depressed body weight, and semen production was lowered for recessive white males. Asmundson and Abbott (1961) observed that a sex-linked dominant gene for slow feathering had no effect on hatchability but it reduced growth, especially in females.

CONCLUDING REMARKS

A few concluding remarks will be made concerning possible utilization of single genes in breeding programs.

Favorable situations. Improving economic performance with the aid of Mendelian factors is a case of indirect selection. Such selection is helpful when no genetic gain is expected from direct selection ($\Delta G = 0$). ΔG is proportional to selection intensity (i) and heritability of the trait (h^2). Table 20.16 lists situations favoring the application of indirect selection using Mendelian factors.

Single gene effects reported in the literature correspond to situations listed in Table 20.16. They include *Na* improving carcass yield and adaptation to hot temperatures, *B* alleles conferring resistance to specific pathogens, and *dw* in broiler breeding suppressing an unfavorable genetic correlation between growth and adult weight. The use of the *dw* gene in egg production might avoid unwanted correlated responses in long-term selection for reduced body size.

Another requirement is a sufficiently large effect of a single gene on quantitative traits of interest. If conventional selection for production traits tends to plateau, previously neglected single genes may become useful; for instance, for egg production the pea comb gene may improve feed efficiency by two percent.

The effects of a gene ought to be consistent, at least under specified conditions.

Table 20.16. Situations favoring the use of indirect selection. (Modified from Mérat, 1975).

Reasons for use of indirect selection	Examples
(a) $i = 0$ (no selection intensity)	
(a_1) Trait costly and/or difficult to measure	Feed efficiency of laying hens; yield of carcasses; percent cracked eggs (?); laying persistency (?)
(a_2) Environment costly and/or difficult to control	Resistance to specific diseases; adaptation to 'exotic' climates (eg. heat)
(a_3) Presence of unfavorable genetic correlations	Fast chick growth; feed efficiency of dam
(b) $i \neq 0, h^2 = 0$ Nonadditivity of remaining genetic variance (eg. overdominance)	Laying intensity

Frequency of a single gene trait in commercial lines. Increasing the frequency of a gene in a commercial line poses less of a problem if it is already present in it. Conversely the eradication of an undesired gene is relatively simple. The absence of a gene from commercial stocks requires its introduction from populations with usually inferior performance, followed by repeated backcrossing to the improved line. In this respect the easy identification of homozygous and heterozygous genotypes, as for the naked neck gene, is an advantage. Because nearly all commercial stocks are crossbred, the use of a recessive gene in the terminal commercial generation would require presence of the gene in all parental lines. Use of the *dw* gene in broiler crosses is a special situation since *dw* expression is wanted only in the maternal parent.

Optimal utilization of a major gene according to its overall effects and factors conditioning its expression. It is necessary to optimize the use of a given gene for rarely are all of its effects positive, and its expression often depends on the genetic background and/or the environment. This implies that effects of a mutation on *all* the aspects of economic value should be investigated. This need to consider overall balance is not peculiar to Mendelian factors; the Cornish breed does not have exceptional qualities for reproduction, but it is widely used owing to its other advantages.

Examples of variable expression of a potentially useful gene according to genetic background and/or environment have already been mentioned, for instance the effects of the *dw* gene on egg laying and efficiency depending on body weight, and the naked neck gene interacting with ambient temperature for many traits.

Consequently the use of a single gene should be optimized in three ways. First, by limiting it to the genetic background and environment where it is most beneficial. Second, by compensating for undesirable effects associated with the mutant either by using additive effects of other genes or by environmental factors, for instance, by introducing the *dw* gene for egg production into stocks selected for large egg size. And third, by investigating favorable nonadditive effects, for instance joint use of two alleles at the *B* locus each bringing dominant resistance to a different pathogen, use of an interaction between the *dw* and *Na* genes for laying rate and efficiency, or incorporating *dw* into populations which tend to overconsume.

Which classes of genes to investigate. The number of identified genes with potential usefulness at present is limited. A few suggestions for the further discovery of useful genes may be the following. Genes related to any reaction with pathogens are obvious candidates, such as immune response genes, receptors to specific pathogens, endogenous proviral genes, or protein and enzyme variants. More research is needed on enzyme polymorphisms in connection with utilization of specific nutrients containing antinutritional or subtoxic factors; there seems to be more scope for such research than there is for investigating genetic variation in requirements or utilization of essential nutrients. More attention should be paid to the composition of egg and meat products; single mutations affecting proteins and enzymes may be worth considering in this regard. Genes recognized by visible effects have been investigated mainly for their influence on body size and on energy metabolism; perhaps behavior and activity, which influence feed conversion, might be found to be influenced by genes with major effects on neurological or musculoskeletal systems.

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Chapter 21. LINKAGE RELATIONSHIPS AND GENE MAPPING

J. James Bitgood and Ralph G. Somes, Jr.

Knowledge of the linkage relationships of genes is important to both the breeder and the researcher. The most detailed linkage map in the chicken is that of the Z, or sex chromosome, developed through the efforts of many researchers over the past 80 years. When compared to sex chromosome maps of some other species, however, knowledge of the genetic composition of this chromosome is very limited.

The chicken chromosome map has evolved slowly over the 80 years since Spillman (1908) showed that barring was sex-linked. The first map was published by Hutt (1936), followed by revisions by Hutt and Lamoreux (1940), Hutt (1949, 1960, 1964), Etches and Hawes (1973), and Somes (1973, 1978, 1987). The last extensive review of linkage relationships in the chicken was by Etches and Hawes (1973). Other summaries are available (Abbott and Yee, 1975; Somes, 1979; Bloom, 1979; Etches and Hawes, 1979), and an updated map has been published on a recurring basis (Somes, 1988).

Knowing the linkage relationships of genes is important, in that it is easier to plan studies of the interrelationships of these genes. Hutt (1964) stated most succinctly the reasons for linkage studies: "...knowing the linkage relationships of the genes involved, the breeder can predict results, can determine the simplest way to attain his objectives, and can see in advance the scope of the operation necessary to that end." Forsythe (1984, personal communication) stated "...if you don't have a map, you don't know where you are going...". This reasoning is even more apropos in this era of genetic engineering with gene transfer, cell hybridization, cloning, and other genetic and molecular advances. It should be easier to manipulate genes once their location is known.

There have been enormous advances in molecular biology in recent years, with an increased potential for genetic manipulation in avian species. Using current genetic techniques, and techniques still being developed and perfected, it may be possible to identify genes or groups of genes that create the superior meat or egg type parent in breeding flocks.

Standardized gene nomenclature was developed for poultry genetics at a symposium during the 66th Annual Meeting of the Poultry Science Association (Somes, 1980). This has aided researchers in identifying new mutations, and has eased the difficulty sometimes found in literature searches. Included in the standardization was the symbol *Ea* (erythrocyte alloantigen) for the avian blood groups. This helped correct confusion that had existed for several years in this field.

Symbolism used in identifying linkage groups and respective chromosomes is more standardized since the review by Etches and Hawes (1973). Most avian geneticists now number the chromosomes in descending order of length, with the Z and W chromosomes unnumbered. This was recommended by Etches and Hawes (1973) and by the St. Paul Conference on Standardization of Avian Chromosome Nomenclature in 1980 (see Chapter 19) and is the method used in this review.

Etches and Hawes (1973) suggested that the then newly developed lines of chromosome rearrangements could be useful in chromosome mapping. This prediction has proven to be true. After Zartman (1973) used chromosome rearrangement break points (R_Bs) to assign the pea comb locus to chromosome 1, other loci were mapped in relation to other R_Bs. The centromere of chromosome 1 was mapped in relation to pea comb and blue egg shell color by using R_{BS} (Bitgood, 1985a). Fittingly enough, this mapping used one of the translocations that Zartman (1973) employed in his pioneering report assigning *P* to chromosome 1.

In this chapter changes to the chicken chromosome map reported since the revision by Etches and Hawes (1973) will be reviewed, and available linkage information on the other species of interest to this book will be summarized.

SEX CHROMOSOMES OF CHICKENS

Etches and Hawes (1973) and the St. Paul Conference on Standardization of Avian Chromosome Nomenclature in 1980 (see Chapter 19) recommended that neither of the linkage groups on the Z (former linkage group V) nor the W (former linkage group VI) chromosomes be assigned a number. This follows procedures used in other species and is adopted in this review. It is further recommended that to prevent possible confusion the numbers V and VI not be assigned to any future linkage group.

The W chromosome (former linkage group VI). Several investigators (Gilmour, 1967; Bacon and Craig, 1966, 1969) presented evidence for a W-linked histocompatibility locus. McCarrey et al. (1981) offered evidence that the H-W antigen is female determining in birds. Mueller et al. (1979) induced expression of the H-W antigen in embryonic ZZ males by estrogen treatment. This would occur only if the H-W structural gene was present in both sexes. There apparently is such a locus but the location remains in question. The finding of a homologous pairing region on the Z and the W chromosomes (Solari, 1977) suggests that there may be loci in this region. The H-W locus could be located here, normally being activated only in the female. Genes apparently exist in this homologous region. The gene for the enzyme muscle creatine kinase in the Harris' hawk is located on both the Z and the W chromosome (Morizot et al., 1987). Studies with other avian species are needed.

The W chromosome contains two repeating DNA units, comprising about 46 percent of the DNA in this chromosome (Tone et al., 1982, 1984). One unit is .6 kb in length, repeated about 14,000 times. The other is 1.1 kb in length, repeated about 6,000 times. These sequences are also found in three species of junglefowl (*G. lafayettei* was not tested), but not in the Japanese common pheasant, guinea fowl, Japanese quail, or the rock dove. The authors speculated that the size, organization, and the numbers of these repeating units, rather than the DNA sequences themselves, might be important in the heterochromatization of the W chromosome. One species of junglefowl, *G. varius*, produced a different Msp I digestion pattern than two other junglefowl species tested. This may be additional evidence that this species is more distantly related to the domestic fowl than are the other species of junglefowl.

The Z chromosome (former linkage group V). Hutt (1964) had assigned 13 loci to the Z chromosome. It was later determined that two of these were alleles at other loci. These were *Sd* (sex-linked dilution), an allele at the barring locus (*B*), and *al* (sex-linked albinism), an allele at the silver locus (*S*). These changes were incorporated into the map of Etches and Hawes (1973), which included 14 loci. Prenatal (*pn*), wingless (*wg*), and liver necrosis (*ln*) were added to Hutt's map. Eleven additional loci were listed as being assigned to this chromosome but not mapped. Maps published by Somes (1973) and Abbott and Yee (1975) were similar to the above two maps.

It is conventional when presenting karyotypes (chromosome displays) to orient each chromosome such that the short (p) arm is at the top, and the long (q) arm is at the bottom (Eldridge, 1985). It is therefore logical to present linkage maps in the same manner. After a series of tests using cytogenetic techniques not available to previous researchers, it became apparent that the published linkage map of the Z chromosome did not meet these criteria. The first of these studies was by Telloni et al. (1976), who used a Z-linked chromosome translocation to study linkages. They reported that the rearrangement break point (R_B) of a t(Z;micro) translocation, the OH t(10), was linked to the feathering locus (*K*) by 23 map units (MU), and segregated independently from barring. Kaelbling and Fechheimer (1983) measured lengths of synaptonemal complexes formed in this translocation. The R_B is on the short arm of the Z chromosome, and therefore *K* is on the short arm.

The MN t(Z;1) R_B (Wang et al., 1982) is 22 MU from *B*, and the NM 7092 t(Z;1) R_B (Zartman, 1973) is six MU from *S* (Bitgood et al., 1980). *S* and *K* are closely linked, so the NM 7092 R_B is on the short arm of the Z chromosome. Bitgood (1980) conducted a G-band analysis of these two R_Bs and found that the MN t(Z;1) R_B was on the long arm marked by a terminal large G-band light staining region, and the NM 7092 t(Z;1) R_B is on the opposite short arm.

Bitgood and Whitley (1986) reported that the locus for popeye, a chicken Z-linked keratoglobus, is on the short arm and linked with *K* and the NM 7092 t(Z;1). Results suggested a linear order of *pop* - R_B - (*S*,*K*). The direction of the centromere is unknown.

Bitgood (1985b) tested the R_B of the MN t(Z;3) against *S*. No recombinants were recovered among 217 backcross progeny. Wang and Shoffner (1974) had shown that this R_B was on the chromosome arm not marked by the terminal large light staining G-band region. Bacon et al. (1985) reported that *ev21* is closely linked to *K*. As *S* is closely linked to *K*, these four markers (the MN t(Z;3) R_B, *ev21*, *S* and *K*) are all located in an extremely small segment of the short arm.

These studies have permitted partial correlation of the linkage map and the cytological map of the chicken Z chromosome and resulted in a change in the orientation of the map. The latest maps (Figure 21.1; Somes, 1987) are inverted when compared to previous maps. The loci for restricted ovulator (an addition to the map), naked and shaker are now displayed at the top, with head streak and barring at the bottom of the map. Only the orientation and not the linear relationships within the map are affected.

In other studies, McGibbon (1981) reported a Z-linked recessive white skin mutation (*y*). Bitgood (1988) tested the linear relationships of *B*, dermal melanin inhibitor (*Id*), *y* and the MN t(Z;1) R_B. The order is *B*, *Id*, R_B, *y*, with *y* closest to the centromere.

Linkage maps have shown the distance between *B* and *Id* as ten, apparently based on Punnett's (1940) comment that this distance "...be regarded as somewhere around ten." Serebrovsky and Petrov (1930) had reported 16 ± 5 map units between these loci. Bitgood (1988) examined 263 testcross progeny and found linkage of 13.7 ± 2.2, so it is probable that the true distance between these markers is in the vicinity of 14 rather than ten. This value is used in Figure 21.1.

Shoffner et al. (1982) reported a protoporphyrin mutation (*pr*) associated with the R_B of the NM 7092 t(Z;1) that eliminates almost all egg shell pigmentation. It is inherited as a Z-linked trait because of the Z-linked nature of the translocation. It is unknown if the R_B affects a gene on the Z chromosome or chromosome 1.

Four Z-linked dominant agglutinogen loci have been reported. Pw_1 allows pokeweed seed extracts to agglutinate red blood cells. Pw_2 allows agglutination only in sexually mature pw_1/pw_1 females (Mizutani et al., 1984). St_1 allows potato rhizome extract to agglutinate red blood cells. St_2 allows agglutination only in sexually mature st_1/st_1 females (Mizutani et al., 1979, 1980).

Several loci have had additional alleles assigned. McGibbon (1977a) reported a fourth allele at the *K* locus, K^s, with the order of dominance $K^n > K^s > K > k^+$.

McGibbon (1974, 1978) reported two additional alleles at the *Id* locus. One was id^c, found in randombred Cornell controls. The second was id^a, found in a strain of Anconas. They were not tested for dominance relationships.

Two alleles were added to the dwarf locus, Macdonald dwarfism (dw^M) (Macdonald College of McGill University) (Hsu et al., 1975), and bantam dwarfism (dw^B) (Custodio and Jaap, 1973). It is not known if they are different alleles or what the order of dominance might be.

Three loci have had linkage relationships reported. Sheridan (1979) found linkage of 4.2 MU between ladykiller (*lk*) and *K*, with *lk* on the opposite side of *K* to *dw* and prenatal (*pn*). This places *lk* toward the centre of the chromosome. Restricted ovulator (*ro*) mapped 33 MU from *K*, independent of *Id* (McGibbon, 1977b), placing it on the short arm of the chromosome. The recessive congenital baldness locus (*ba*) (Somes, 1970) was mapped 7.6 ± .03 map units from the s^{al} locus (Somes, unpublished). No other mapping tests were conducted.

Two viral loci have been assigned to the Z chromosome. A defective ALV provirus, *ev7* (Smith and Crittenden, 1981), is near the end of one of the chromosome arms (Tereba et al., 1981). An endogenous retrovirus, *ev21* (Bacon et al., 1985), is closely linked to the *K* locus.

A map of the chicken Z chromosome is shown in Figure 21.1. Loci that have been assigned to the Z chromosome but have not yet been mapped are shown in Figure 21.2.

AUTOSOMAL LINKAGE IN CHICKENS

Chromosome 1 (linkage group III). Hutt (1964) showed six loci in this linkage group. He stated that Warren (1938) had previously assigned three of these loci (*Na*, *h* and *Fl*) to a separate group. Etches and Hawes (1973) showed ten loci mapped in this group, with three additional loci assigned but unmapped. Based on their review of the literature, they indicated that *Na*, *h* and *Fl* were part of this group.

The assignment of pea comb (*P*) to chromosome 1 (Zartman, 1973) resulted in the first assignment of a chicken linkage group to an autosome. Since then a number of studies have been conducted on linkage relationships on this chromosome. Some studies have provided additional clarification of the relationships, and others have contributed to the current confusion.

Following Zartman's (1973) report, Bitgood et al. (1980) reported results of tests between several chromosome rearrangement break points (R_B) and several morphological markers. *P* showed linkage to the MN inv(1) and the MN t(1;micro), both R_B's on the short arm. *P* and blue egg (*O*) were linked to the MN t(Z;1) R_B on the short arm of the chromosome. The order was R_B - *O* - *P*, with *P* closest to the centromere of the chromosome. Bitgood (1985a) tested the NM 7659 t(Z;1) (Zartman, 1973) against *O* and *P*. This R_B is on the long arm, proximal to the centromere. This test showed that the linear order was R_B - *P* - *O*. Both sets of results indicate that the linear order of these markers is

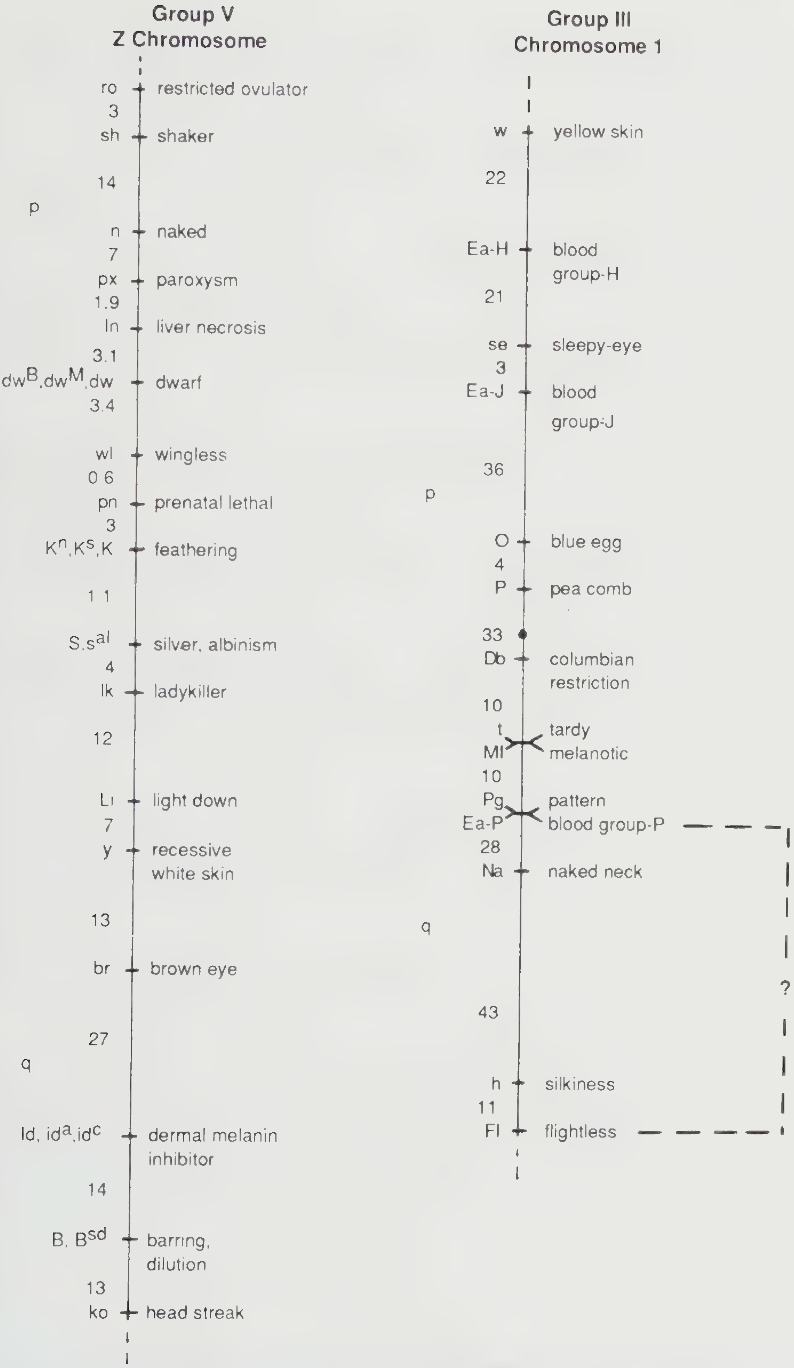

Figure 21.1. Linkage maps of the chicken Z chromosome and chromosome 1. The dotted line for chromosome 1 indicates questionable assignments.

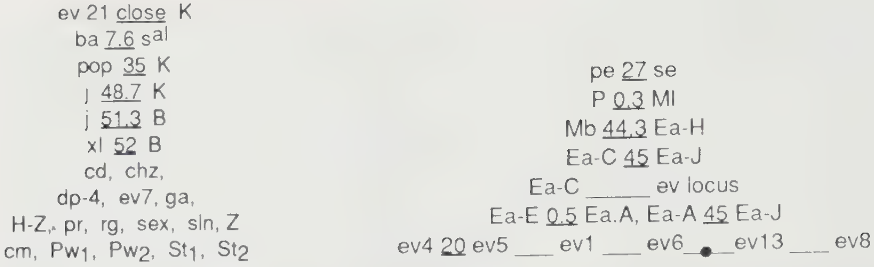

Figure 21.2. Loci assigned to the Z chromosome (right) and to chromosome 1 (left) but not yet mapped. The six *ev* loci of chromosome 1 would be good markers to test against morphological traits to further develop the map. The centromere is between *ev6* and *ev13; ev13* and *ev8* are on the short arm.

NM 7659 t(Z;1) - centromere - *P* - *0* - MN t(Z;1).

In the test with the NM 7659, *P* and *0* showed 3.1 ± 1.1 map units, on the arm not containing the R_B. Recombination should not have been affected by the translocation. Bitgood et al. (1983) reported 4.28 ± 1.3 map units between these loci. Crawford (1986) found 2.39 ± 8.54 map units between these loci. The previous map estimate of five map units was based on two recombinants among 35 individuals (Bruckner and Hutt, 1939). As all the above tests show less than five map units, the linkage estimate in the current map is revised to four map units (Figure 21.1).

Briles et al. (1967) reported that yellow skin (*w*) and blood group *H* (*Ea-H*) were linked by 22 map units. Bitgood et al. (1980) reported 17 map units between these same loci. Somes (1968) found 45 percent recombination between *P* and sleepy eye (*se*) and 43 percent recombination between *se* and *w*. Etches and Hawes (1973) suggested the order of loci in this region is *P* - *0* - *se* - *Ea-H* - *w*, placing these loci on the short arm. Bitgood et al. (1980) tested *w* against three R_Bs on this arm, and *Ea-H* against one of these R_Bs, with no evidence of linkage. These R_Bs are all closely linked to *P*. Because of the long length of the chromosome, it is possible that loci on the same arm will show independent assortment. These results do not rule out the assignment of the loci for *w* and *Ea-H* to this chromosome.

Bitgood et al. (1987b) confirmed previous suggestions that tardy feathering (*t*) was part of this linkage group. By testing *t* against *P* and the MN t (Z;1) rearrangement, the authors showed that the probable order is R_B - *P* - centromere - *t*, with *t* on the long arm.

Crawford (1986) reported that the plumage color mutation melanotic (*Ml*) was .3 map units from *P* and 2.5 map units from *0*. He suggested the order of centromere - *Ml* - *P* - *0*. The evidence looked attractive until Carefoot (1987b) reported 46 map units between *Ml* and *P*. Because of the similarity in the phenotypes described in the two reports, more studies are needed to clarify this. With the number of plumage pattern mutations reported in the chicken and the different effects created by various genetic backgrounds, it would not be surprising if Carefoot (1987b) and Crawford (1986) were studying two different mutations with similar effects.

Washburn and Smyth (1967) reported that a gene later shown to be dark brown (*Db*) (Smyth and Malone, 1979) was linked to *P* by 28 map units. Moore and Smyth (1972) reported that *Db* was linked to autosomal barring (*Ab*) by 17 map units. Carefoot (1985, 1986) reported that lacing (*Lg*), *Ab* and pencilling (*Pg*) were all the same gene; expression depends on the allele present at the *Ml* locus. He recommended that the gene symbol *Pg* (patterning) be used for this locus. Carefoot (1987a, 1987b) reported linkage of ten map units between *Db* and *Ml*, and 46 between *Ml* and *P*, with *Ml* between *Db* and *Pg*.

The Etches and Hawes (1973) map assigned marbled down (*ma*) reported by Hertwig (1933) to this chromosome, linking it to *P* by 33 map units. Carefoot (1988, personal communication) conducted a series of studies and concluded that *Db* in the presence of *E* (extended black) and *S* created the *ma* phenotype. Phenotypic descriptions and linkage distance from *P* support this hypothesis. *Db* replaces *ma* in Figure 21.1. The gene order in this region is *P - Db - Ml - Pg*.

The assignment of naked neck (*Na*), silky feathering (*h*) and flightless (*Fl*) to this chromosome has been a subject of discussion for a long time. Hutt (1949) stated that Hertwig (1933) had reported 32.8 percent crossing over between *ma* and *P*, and 45.6 percent between *ma* and *Na*. Warren (1938) reported 43 percent crossing over between *Na* and *h*, and placed *Na*, *h* and *Fl* in a separate linkage group. Hutt (1949) placed *Na* in the *0 - P - ma* group, and showed *h* and *Fl* in a separate group.

Hutt (1964) assigned all of these loci to one linkage group, but included a dotted line and a question mark between *ma* and *Na*. This restriction was followed by Somes (1973). Etches and Hawes (1973) removed the dotted line and question mark, and maps since then have followed this format.

Na is linked to *Ea-P* (Briles et al., 1967; Bitgood et al., 1980, 1984). Pooled data from the latter two reports indicate linkage of 27.9 ± 2.3. The map in Figure 21.1 shows 28 map units. Several studies have tested both of these loci against other morphological traits and several chromosome rearrangements reported to be on chromosome 1 (Bitgood et al., 1980, 1983, 1984). No linkage was evident in any of the tests.

Silky feathering (*h*) has been tested against *Na* and *Ea-P* with no evidence of linkage (Bitgood et al., 1984). In another study (Kendall and Tsai, 1988, personal communication) *t* and *h* were tested using 397 progeny from an intercross mating with no evidence of linkage.

To indicate that assignment of these loci to this chromosome is somewhat questionable, a dotted line and a question mark are used in Figure 21.1, similar to the annotation used by Hutt (1964). Firm evidence of different linkage relationships will be required to delete these loci from the linkage group.

The major difficulty in studying linkage relationships in this linkage group is its size. The inability to identify intervening loci to tie confirmed sections together retards progress. Loci that are more easily classified and more closely linked are needed to construct a truly reliable map.

Several defective ALV proviral loci have been assigned to chromosome 1: *ev1* (Tereba and Astrin, 1980), *ev4*, *ev5* and *ev8* (Tereba and Astrin, 1982), and *ev6* (Tereba, 1981). An endogenous retrovirus (*ev13*) has been assigned to this chromosome (Tereba and Astrin, 1982). These assignments were made by *in situ* hybridization procedures, using viral RNA as a probe (reviewed by Tereba, 1983). From the distal end of the long arm, the order is *ev4-ev5-ev1-ev6-*(centromere)*-ev13-ev8*. These have not been tested against other loci for linkage relationships.

Hughes et al. (1979) reported that the ß-type globin genes were on either chromosome 1 or 2.

Loci that have been assigned to chromosome 1 but not yet placed on the map are shown in Figure 21.2.

Chromosome 2. Presumably this chromosome contains linkage group I, II or IV, but none of the loci in these groups has been mapped.

A limb mutation, shankless (*shl*), is tightly linked to a pericentric inversion on chromosome 2 (Langhorst and Fechheimer, 1985).

The *ev2* locus has been assigned to the long arm of this chromosome (Tereba et al., 1981).

The ß-actin gene is located either on the long arm of chromosome 2 or on a chromosome in the size range of 9-12 (Shaw et al., 1988). Both sites were labelled after *in situ* hybridization studies. ß-actin is part of a multigene family, and one of these locations may contain one of the other actin genes.

In situ hybridization has assigned two oncogenes to chromosome 2. These are *c-erb-B* (Symonds et al., 1984) and *c-myc* (Symonds et al., 1986).

The ß-type globin genes and the ovalbumin gene (*Ov*) are on either chromosome 2 or 3 (Hughes et al., 1979). Ovoglobulin G_3 (G_3) and *Ov* are linked by seven map units (Durand and Mérat, 1982), so G_3 is also on one of these chromosomes.

Linkage groups I, II and IV. These groups have changed little since the map published by Hutt (1964). In the review by Etches and Hawes (1973), *Mp* and *lav* were added to linkage group I. Neither locus has been mapped (Figure 21.3).

Ziehl and Hollander (1987) reported a new allele at the *I* locus (linkage group II, Figure 21.3). Dun (I^D) is incompletely dominant, affecting only eumelanin. It creates a dun pattern when heterozygous. Dun is described as a brownish or neutral brownish grey or dull greyish brown. The homozygote is red-pyle or near-white. This is the only change to the Etches and Hawes (1973) map in linkage group II.

Somes (1986) reported that buttercup comb and V-shaped comb were two alleles at the duplex comb locus on linkage group IV (Figure 21.3). The buttercup comb was assigned the gene symbol D^C (for cup) and the V-shaped comb found in breeds such as the Polish was assigned the gene symbol D^V (for V-shaped). D^V is dominant to D^C. This is the only change to the Etches and Hawes (1973) map in linkage group IV.

Some loci have been assigned to chromosome 2, as discussed earlier. Presumably this chromosome also contains one of the three linkage groups described above.

Linkage groups VII through X. Kao (1973) mapped adenine synthesis A (*Ade-A*) to chromosome 6, and adenine synthesis B (*Ade-B*) to chromosome 7. Little standardization existed at that time and linkage maps numbered the Z chromosome as 5. Kao's chromosomes 6 and 7 were shown as 7 and 8 on subsequent maps. Following agreement at the St. Paul Conference on Standardization of Avian Chromosome Nomenclature in 1980 (see Chapter 19) the Z chromosome is no longer numbered. Linkage group VII is on chromosome 6 (Figure 21.3), and linkage group VIII is on chromosome 7 (Figure 21.3). Kao's original designations of chromosomes 6 and 7 are used here.

Palmer and Jones (1986) used somatic cell hybridization to assign three genes, phosphoglucomutase (*Pgm-2*), serum albumin (*Alb*), and vitamin D binding protein (*Gc*), to chromosome 6 (linkage group VII, Figure 21.3). They verified the assignment of phosphoribosyl pyrophosphate amidotransferase (PPAT) to this chromosome. It was originally assigned by Kao (1973), using the nomenclature of *Ade-A* (linkage group VII). Close linkage between *Gc* and *Alb* was demonstrated by Juneja et al. (1982). This is an example of linkage group conservation, because linkage of these genes has been reported in a number of species. Linkage information from one species can be a guide in planning studies in other species.

Bloom and Bacon (1985) showed that the nucleolar organizing region (NOR) and the major histocompatibility complex (MHC) in the chicken are on a microchromosome

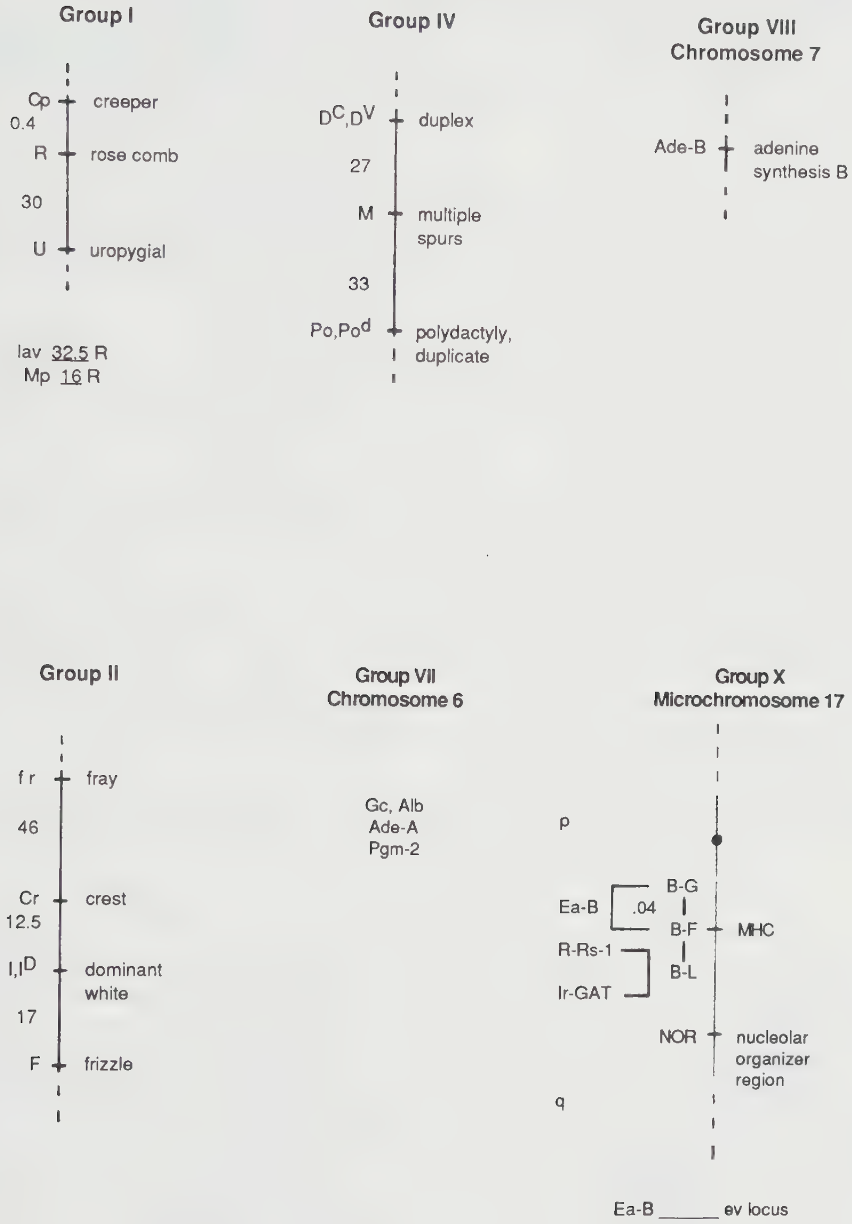

Figure 21.3. Maps for chicken linkage groups I, II, IV, VII, VIII, and X. Three of the linkage groups have been assigned to specific chromosomes. The suborder within the MHC of group X (microchromosome 17) relative to the NOR is not known.

(linkage group X, Figure 21.3). Auer et al. (1987) assigned the NOR to chromosome 17. This also places the MHC on this chromosome.

It is recommended that reference to linkage group IX be discontinued. This was not a classical linkage group, but was convenient for listing loci found on microchromosomes. It is further recommended that the designation IX not be used in the future to prevent possible confusion. Loci that have been assigned to microchromosomes are listed in Table 21.1.

Other linkage relationships. Brumbaugh et al. (1983) reported that autosomal albinism (*a*) (Warren, 1933) was an allele at the recessive white locus (*c*), and recommended use of the gene symbol c^a. This may not be the same as Warren's albinism, because Warren reported independent segregation between *c* and *a*. Smyth et al. (1986) reported a fourth allele at this locus, describing a red-eyed white mutation, with gene symbol c^{re}. The order of dominance is $C^+ > c > c^{re} > c^a$.

Bitgood et al. (1980) reported possible linkage of 40.0 ± 4.2 between yellow skin (*w*) and a feathered shank gene. This agreed with Lambert and Knox (1929) who found 43.3 percent recombination between *w* and a feathered shank phenotype. Serebrovsky (1926) suggested the existence of at least two dominant and at least two recessive genes that created feathered shank phenotypes.

Shaw et al. (1988) reported that *in situ* hybridization using a probe for the ß-actin gene labelled the long arm of chromosome 2 and a chromosome in the size range 9-12. As ß-actin is apparently a single gene in the chicken, one of these sites might be another gene in the actin multigene family.

Several loci have been mapped to chromosome 3. Symonds et al. (1984) reported that *c-myb* is on this chromosome. However Tereba and Lai (1982) had reported that this locus was on a microchromosome. Tereba et al. (1981) mapped *ev14* to this chromosome. Hughes et al. (1979) reported that ovalbumin (*Ov*) is on either chromosome 2 or 3. Durand and Mérat (1982) reported linkage of seven map units between ovoglobulin G_3 and *Ov*, so G_3 is also on either chromosome 2 or 3.

Linkage relationships between several loci have been reported, but these loci have not been assigned to a chromosome or to a linkage group (Table 21.2).

NEGATIVE LINKAGE TESTS IN CHICKENS

A phenomenal storehouse of negative results that have not been published probably exists in laboratory files throughout the world. Negative results should be published or in some other way made available to investigators. This would help prevent unnecessary duplication of effort.

A listing of tests that have been conducted with no linkage being found between loci is given in Table 21.3. This is a modification of the list by Etches and Hawes (1979) including tests conducted since that review was published.

Table 21.4 lists published tests that have shown independent segregation between chromosome rearrangement break points and several loci. Rearrangement nomenclature is adapted from ISCN (1978). The symbols p (short arm) and q (long arm) designate the chromosome arm involved, and the symbols + or - describe the morphological change. As an example 1p+ indicates that the break point is on the short (p) arm of chromosome 1 and the arm has been lengthened (+) by the addition of a segment of DNA from another chromosome.

Table 21.1. Loci on microchromosomes.

Gene symbol	Trait name	Reference
c-erb-A	Cellular oncogene of avian erythroblastosis virus A	Symonds et al. (1984)
c-ets	Cellular oncogene of avian E-26 leukemia virus	Symonds et al. (1986)
c-fps	Cellular oncogene of avian Fujinami sarcoma virus	Symonds et al. (1984)
c-mil/mht	Cellular oncogene of avian Mill Hill 2 leukemia virus	Symonds et al. (1986)
c-myb	Cellular oncogene of avian myelablastosis virus	Tereba and Lai (1982)
c-src	Cellular oncogene of Rous sarcoma virus	Tereba et al. (1979)
ev3	Defective ALV provirus, gs, chf$^+$, V$^-$	Tereba (1981)
--[1]	α-type globin genes	Hughes et al. (1979)
Hprt	Hypoxanthine phosphoribosyltransferase	Rasko et al. (1979)
Ovm	Ovomucoid	Hughes et al. (1979)
Tf	Transferrin	Hughes et al. (1979)
Tk-F	Cytosol thymidine kinase F	Leung et al. (1975)

[1] No gene symbol assigned.

Table 21.2. Unmapped linkage relationships.

Symbols and distance	Trait names	Reference
Ea-M(5.4)*Ea-Q*	Erythrocyte alloantigen M - erythrocyte alloantigen Q	Scheinberg (1956)
Ea-O(2.6)*Ea-S*	Erythrocyte alloantigen O - erythrocyte alloantigen S	Scheinberg (1956)
Es-1(.04)*Es-2*	Esterase-1 - esterase 2	Kimura and Kameyama (1970)
IgG-1(2.8)*IgM-1*	7S-1 immunoglobulin heavy chain - immunoglobulin M	Derka and Hala (1979); Pink and Ivanyi (1975)
Tv-A(.17)*Tv-C*	ALV subgroup A - ALV subgroup C	Dren and Pani (1977); Pani (1974)
Tv-B(close)*Tv-E*	ALV subgroup B - ALV subgroup E	Robinson and Lamoreux (1976)
--[1]	δ subunit - γ subunit of nicotine acetylcholine receptor	Nef et al. (1984)
--[1]	δ 1-Crystallin - δ 2-Crystallin	Hann (1966)

[1] No gene symbols assigned.

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979).

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
a	Cr	9	am	Na	9	con A	am	10	Cr	w	9
	D	9		P	9						
	F^1	9		Po	9	Cp	Cr	9	D	a	9
	Fl	9		R	9		D	9		am	9
	Mb	9		w	9		F	9		Bl	9
	Na	9					I	9		c	9
	P	9	As	Db	9		Mb	9		Cp	9
	Po	9		P	9		P	9		Cr	9
	R	9					Po	9		E	9
	Rp	9	B	S^2	6		Rp	9		F	9
	w	9		y^2	2		se	9		Fl	9
							w	9		fr	9
Acp-2	Akp	9	bc	rc	7					Hf	12
	c	9				Cr	a	9		I	9
	Ea-A &		Bl	Cr	9		am	9		Mb	9
	Ea-E	9		D	9		Bl	9		mo	9
	Ea-B	9		F	9		c	9		Mp	9
	Ea-D	9		Fl	9		Cp	9		Na	9
				fr	9		D	9		P^1	9
Akp	Acp-2	9		h	9		Ea-A &			pk	9
	c	9		P^{D3}	13		Ea-E	9		Po^1	9
	Ea-A &			Mb	9		Ea-B	9		R	9
	Ea-E	9		Na^1	9		Ea-C	9		ropy	9
	Ea-B	9		P	9		Ea-D	9		Rp	9
	Ea-D	9		Po	9		Ea-H	9		se	9
	Es-1	9		R^1	9		Ea-I	9		sn	9
				ropy	9		Ea-J	9		sw	9
Alb	Ea-A &			Rp	9		Ea-K	9		t	9
	Ea-E	9		w	9		Ea-L	9		w^1	9
	Ea-B	9					Ea-P	9			
	Ea-C	9	c	Acp-2	9		epi	9	Db	As	9
	Ea-D	9		Akp	9		Fl	9			
	G_2	9		Cr	9		Hf	12	dehy	dp-1	9
	G_3	9		D	9		M	9		eu	9
	I	9		Ea-P	3		Mb	9		sc	9
	IgM-1	9		F	9		mo	9			
	Mr-1	9		Fl	9		Na	9	dp-1	dehy	9
	P	9		h	3		P	9		dp-2	9
	Pa	9		Hf	12		pk	9		dp-3	9
	Tf	9		Mb	9		Po	9		eu	9
	w	9		Na	3, 9		R	9		ta-2	9
				P	9		ropy	9			
am	con A	10		pk	9		Rp	9	dp-2	dp-1	9
	Cr	9		Po	9		sc	9			
	D	9		R	9		se	9	dp-3	dp-1	9
	F	9		Rp	9		sn	9			
	I	9		U	9		sw	9	dp-5	I	11
	Mb	9		w	9		t	9		Na	11

Continued...

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979). (Continued)

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
dp-5	P	11	Ea-B	Ea-D	9	Ea-D	Cr	9	Ea-I	Ea-J	9
	R	11		Ea-H	9		E	9		Ea-K	9
				Ea-I	9		Ea-A &			Ea-L	9
E	D	9		Ea-J	9		Ea-E	9		Ea-P	9
	Ea-A &			Ea-K	9		Ea-B	9		Mb	9
	Ea-E	9		Ea-L	9		Ea-C	9		Na	9
	Ea-B	9		Ea-P	9		Ea-H	9		P	9
	Ea-C	9		G_2	9		Ea-I	9		Po	9
	Ea-D	9		G_3	9		Ea-J	9		R	9
	I^{D3}	13		I	9		Ea-K	9		w	9
	P	9		IgM-1	9		Ea-L	9			
	Po	9		Mb	9		Ea-P	9	Ea-J	Cr	9
	se	9		Mr-1	9		G_2	9		Ea-A &	
				Na	9		G_3	9		Ea-E	9
Ea-A & Ea-E				Ov	9		Mb	9		Ea-B	9
	Acp-2	9		P	9		Na	9		Ea-C	9
	Akp	9		Po^l	9		Ov	9		Ea-D	9
	Alb	9		R	9		P	9		Ea-H	9
	Cr	9		w	9		Po	9		Ea-I	9
	E	9					R	9		Ea-K	9
	Ea-B	9	Ea-C	Alb	9		w	9		Ea-L	9
	Ea-C	9		Cr	9					Ea-P	9
	Ea-D	9		E	9	Ea-E[4]	Mr-1	9		Mb	9
	Ea-H	9		Ea-A &						Na	9
	Ea-I	9		Ea-E	9	Ea-H	Cr	9		P^l	9
	Ea-J	9		Ea-B	9		Ea-A &			Po	9
	Ea-K	9		Ea-D	9		Ea-E	9		R	9
	Ea-L	9		Ea-H	9		Ea-B	9		w	9
	Ea-P	9		Ea-I	9		Ea-C	9			
	G_2	9		Ea-J	9		Ea-D	9	Ea-K	Cr	9
	G_3	9		Ea-K	9		Ea-I	9		Ea-A &	
	I	9		Ea-L	9		Ea-J	9		Ea-E	9
	Mb	9		Ea-P	9		Ea-K	9		Ea-B	9
	Na	9		G_2	9		Ea-L	9		Ea-C	9
	Ov	9		G_3	9		Ea-P	9		Ea-D	9
	P	9		I	9		Mb^l	9		Ea-H	9
	Po	9		Mb	9		Na	9		Ea-I	9
	R	9		Mr-1	9		P^l	9		Ea-J	9
	w	9		Na	9		Po	9		Ea-L	9
				Ov	9		R	9		Ea-P	9
Ea-B	Acp-2	9		P	9					Mb	9
	Akp	9		Po	9	Ea-I	Cr	9		Na	9
	Alb	9		R	9		Ea-A &			P	9
	Cr	9		w	9		Ea-E	9		Po	9
	E	9					Ea-B	9		R	9
	Ea-A &		Ea-D	Acp-2	9		Ea-C	9		w	9
	Ea-E	9		Akp	9		Ea-D	9			
	Ea-C	9		Alb	9		Ea-H	9			

Continued...

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979). (Continued)

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
Ea-L	Cr	9	Es-1	Akp	9	fr	Bl	9	Hf	I	12
	Ea-A &						D	9		Na	12
	Ea-E	9	eu	dehy	9		F	9		P	12
	Ea-B	9		dp-1	9		I^1	9		Po	12
	Ea-C	9					Mb	9		R	12
	Ea-D	9	F	a^1	9		Na	9		sc	12
	Ea-H	9		am	9		P	9		se	12
	Ea-I	9		Bl	9		Po	9			
	Ea-J	9		c	9		R	9	I	Alb	9
	Ea-K	9		Cp	9		Rp	9		am	9
	Ea-P	9		D	9		w	9		Cp	9
	Mb	9		epi	9	G_2	Alb	9		D	9
	Na	9		Fl	9		Ea-A &			dp-5	11
	P	9		fr	9		Ea-E	9		Ea-A &	
	Po	9		Hf	12		Ea-B	9		Ea-E	9
	R	9		lav	9		Ea-C	9		Ea-B	9
	w^1	9		M	9		Ea-D	9		Ea-C	9
				Mb	9		G_3	9		epi	9
Ea-P	c	3		Na	9		P	9		Fl	9
	Cr	9		pk	9		Pa	9		fr^1	9
	Ea-A &			Po	9		Tf	9		h	9
	Ea-E	9		R	9		w	9		Hf	12
	Ea-B	9		ropy	9					IgM-1	9
	Ea-C	9		Rp^1	9	G_3	Alb	9		M	9
	Ea-D	9		se	9		Ea-A &			Mb	9
	Ea-H	9		sw	9		Ea-E	9		Mp	9
	Ea-I	9		w	9		Ea-B	9		Na	9
	Ea-J	9					Ea-C	9		P	9
	Ea-K	9	Fl	a	9		Ea-D	9		pk	9
	Ea-L	9		Bl	9		G_2	9		Po	9
	Mb	9		c	9		P	9		R	9
	P	9		Cr	9		Pa	9		Rp	9
	Po	9		D	9		Tf	9		se	9
	R	9		F	9		w	9		sn	9
	w^1	9		I	9					sw	9
				M	9	h	Bl	9		t	9
ec	sc	9		Mb	9		c	3		Tf	9
	ta-2	9		mo	9		I	9		w	9
				Na^1	9		Mb	9			
epi	Cr	9		P^1	9		Po	9	I^{D3}	Bl	13
	F	9		pk	9		R	9		E	13
	I	9		Po	9		Rp	9		M	13
	Na	9		R	9		w	9		P	13
	P	9		Rp	9					pk	13
	Po	9		t	9	Hf	c	12		w	13
	R	9		w	9		Cr	12			
							D	12	IgG-1	IgG-2	9
Er	R	9	Fm	sc	9		F	12		IgG-3	9

Continued...

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979). (Continued)

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
IgG-2	IgG-1	9	Mb	F	9	Na	Ea-C	9	P	Cp	9
	IgG-3	9		Fl	9		Ea-D	9		Cr	9
				fr	9		Ea-H	9		D^1	9
IgG-3	IgG-1	9		h	9		Ea-I	9		E	9
	IgG-2	9		I	9		Ea-J	9		Ea-A &	
				lav	9		Ea-K	9		Ea-E	9
IgM-1	Alb	9		M	9		Ea-L	9		Ea-B	9
	Ea-B	9		mo	9		epi	9		Ea-C	9
	I	9		Na	9		F	9		Ea-D	9
	Mr-1	9		O	1		Fl^1	9		Ea-H	9
				P	1, 9		fr	9		Ea-I	9
lav	F	9		pk	9		Hf	12		Ea-J	9
	Mb	9		Po	9		I	9		Ea-K	9
				R	9		I^{D3}	13		Ea-L	9
M	Cr	9		ropy	9		ma	9		Ea-P	9
	F	9		Rp	9		Mb	9		epi	9
	Fl	9		se	9		nk	8		Fl^1	9
	I	9		sw	9		O	5		fr	9
	I^{D3}	13		t	9		P	5, 9		G_2	9
	Mb	9		w	9		pir	4		G_3	9
	mo	9					pk	9		h	9
	P	9	mo	Cr	9		Po	9		Hf	12
	pk	9		D	9		R	9		I	9
	R	9		Fl	9		ropy	9		I^{D3}	13
	ropy	9		M	9		Rp^1	9		M	9
	sw	9		Mb	9		t	9		Mb	1
	w	9		Po	9		w	6, 9		Mp	9
										Na	5
ma	Na	9	Mp	D	9	nk	Na	8		nk	8
				I	9		P	8		Pa	9
Mb	a	9		P	9		R	8		pk	9
	am	9					sc	8, 9		Po	9
	Bl	9	Mr-1	Alb	9					R	9
	c	9		Ea-B	9	O	Mb	1		ropy	9
	Cp	9		Ea-C	9		Na	5		Rp	9
	Cr	9		Ea-E	9					sw	9
	D	9		IgM-1	9	Ov	Ea-A &			Tf	9
	Ea-A &						Ea-E	9		w	6, 9
	Ea-E	9	Na	a	9		Ea-B	9			
	Ea-B	9		am	9		Ea-C	9	Pa	Alb	9
	Ea-C	9		Bl^1	9		Ea-D	9		G_2	9
	Ea-D	9		c	3, 9					G_3	9
	Ea-H	9		Cr	9	P	a	9		P	9
	Ea-I	9		D	9		Alb	9		w	9
	Ea-J	9		dp-5	11		am	9			
	Ea-K	9		Ea-A &			As	9	pir	Na	4
	Ea-L	9		Ea-E	9		Bl	9		shl	4
	Ea-P	9		Ea-B	9		c	9		t	4

Continued...

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979). (Continued)

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
pk	c	9	Po	ropy	9	rc	bc	7	se	Cr	9
	Cr	9		Rp	9					D	9
	D	9		sc	9	ropy	Bl	9		E	9
	F	9		se	9		Cr	9		F	9
	Fl	9		sw	9		D	9		Hf	12
	I	9		t	9		F	9		I	9
	I^{D3}	13		w	9		M	9		Mb	9
	M	9					Mb	9		Po	9
	Mb	9	R	a	9		Na	9		R	9
	Na	9		am	9		P	9			
	P	9		Bl^1	9		Po	9			
	Po	9		c	9		R	9	shl	pir	4
	R	9		Cr	9		Rp	9			
	Rp	9		D	9				sn	Cr	9
	w	9		dp-5	11	Rp	a	9		D	9
				Ea-A &			Bl	9		I	9
Po	a	9		Ea-E	9		c	9			
	am	9		Ea-B	9		Cp	9	sw	Cr	9
	Bl	9		Ea-C	9		Cr	9		D	9
	c	9		Ea-D	9		D	9		F	9
	Cp	9		Ea-H	9		F^1	9		I	9
	Cr	9		Ea-I	9		Fl	9		M	9
	D^1	9		Ea-J	9		fr	9		Mb	9
	E	9		Ea-K	9		h	9		P	9
	Ea-A &			Ea-L	9		I	9		Po	9
	Ea-E	9		Ea-P	9		Mb	9		R	9
	Ea-B	9		epi	9		Na^1	9			
	Ea-C	9		Er	9		P	9	t	Cr	9
	Ea-D	9		F	9		pk	9		D	9
	Ea-H	9		Fl	9		Po	9		Fl	9
	Ea-I	9		fr	9		R	9		I	9
	Ea-J	9		h	9		ropy	9		Mb	9
	Ea-K	9		Hf	12		t	9		Na	9
	Ea-L	9		I	9		w	9		pir	4
	Ea-P	9		M	9					Po	9
	epi	9		Mb	9	S	B^2	6		R	9
	F	9		Na	9		y^2	2		Rp	9
	Fl	9		nk	8					w	9
	fr	9		P	9	sc	Cr	9			
	h	9		pk	9		dehy	9	ta-2	dp-1	9
	Hf	12		Po	9		ec	9		ec	9
	I	9		ropy	9		Fm	9			
	Mb	9		Rp	9		Hf	12	Tf	Alb	9
	mo	9		sc	9		nk	8		G_2	9
	Na	9		se	9		Po	9		G_3	9
	P	9		sw	9		R	9		I	9
	pk	9		t	9					P	9
	R	9		w	9	se	Cp	9		w	9

Continued...

Table 21.3. Genes in the chicken that show independent segregation. References[5] are listed at the end of the table. (Modified and updated from Etches and Hawes, 1979). (Continued)

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
U	*c*	9	*w*	*Ea-A* &		*w*	*F*	9	*w*	*P*	6, 9
				Ea-E	9		*Fl*	9		*Pa*	9
w	*a*	9		*Ea-B*	9		*fr*	9		*pk*	9
	Alb	9		*Ea-C*	9		G_2	9		*Po*	9
	am	9		*Ea-D*	9		G_3	9		*R*	9
	Bl	9		*Ea-I*	9		*h*	9		*Rp*	9
	c	9		*Ea-J*	9		*I*	9		*t*	9
	Cp	9		*Ea-K*	9		I^D[3]	13		*Tf*	9
	Cr	9		*Ea-L*	9		*M*	9			
	D[1]	9		*Ea-P*	9		*Mb*	9	*y*	*B*[2]	2
							Na	6, 9		*S*[2]	2

[1] Possible linkage.

[2] Silver is on the short arm of the Z chromosome. Barring and *y* are on the long arm. Because of long map distances, these three loci show independent assortment when tested against each other.

[3] I^D is allelic to *I* (Ziehl and Hollander, 1987). [4] See also blood group *Ea-A*.

[5] 1-Bitgood, 1985a; 2-Bitgood, 1985b; 3-Bitgood et al., 1980; 4-Bitgood et al., 1983; 5-Bitgood et al., 1984; 6-Bitgood et al., 1986; 7-Bitgood et al., 1987a; 8-Crawford et al., 1982; 9-Etches and Hawes, 1979; 10-Kline and Sanders, 1984; 11-Olympio et al., 1983; 12-Somes et al., 1984; 13-Ziehl and Hollander, 1987.

Table 21.4. Genes showing independent segregation when tested against chromosome rearrangement break points (R_B).

R_B	Gene	Name	Reference
MN t(Z;1)	*Na*	Naked neck	Bitgood et al. (1980)
	Ea-A	Blood group A	Bitgood et al. (1980)
	Ea-E	Blood group E	Bitgood et al. (1980)
	Ea-H	Blood group H	Bitgood et al. (1980)
	Ea-P	Blood group P	Bitgood et al. (1980)
	pir	Pirouette	Bitgood et al. (1987a)
	S	Silver	Bitgood et al. (1980)
	t	Tardy feathering	Bitgood et al. (1987b)
	w	Yellow skin	Bitgood et al. (1980)
NM 7092 t(Z;1)	*P*	Pea comb	Bitgood et al. (1980)
	Na	Naked neck	Bitgood et al. (1980)
	w	Yellow skin	Bitgood et al. (1980)
NM 7659 t(Z;1)	*Id*	Dermal melanin	Bitgood (1985a)
	Mb	Muffs and beard	Bitgood (1985a)
MN t(Z;3)	*B*	Barring	Bitgood (1985b)
	rc	Rods and cones	Cheng et al. (1980)
OH t(Z;micro)	*B*	Barring	Telloni et al. (1976)
MN t(1;4)	*P*	Pea comb	Bitgood et al. (1980)
	w	Yellow skin	Bitgood et al. (1980)

Continued...

Table 21.4. Genes showing independent segregation when tested against chromosome rearrangement break points (R_B). (Continued)

R_B	Gene	Name	Reference
MN t(1;micro)	*Na*	Naked neck	Bitgood et al. (1980)
	w	Yellow skin	Bitgood et al. (1980)
MN inv(1)	*Na*	Naked neck	Bitgood et al. (1980)
	w	Yellow skin	Bitgood et al. (1980)
OH inv(2)	*bc*	Blind, cataracts	Bitgood et al. (1986)
	pir	Pirouette	Bitgood et al. (1987a)

LINKAGE IN OTHER POULTRY SPECIES

Japanese quail. The Japanese quail presents a unique situation. With their short generation interval and large numbers available there should be a well-developed genetic map, but this is not the case. The problem is two fold: a lack of easily described mutations, and few investigators willing to study single gene mutations.

The Z-linked traits reported in Japanese quail are listed in Table 21.5, and the linkage map of the Z chromosome is shown in Figure 21.4. Detailed descriptions of the traits are given in Chapter 13.

There have been three autosomal linkage relationships reported in the quail. Extended brown (*E*) is linked to phosphoglucose isomerase (*Pgi*) (Ito et al., 1988a). A difference in recombination values exists between sexes. Males show 31 percent recombination, and females show 43 percent. Ito et al. (1988b) found 30 percent recombination between panda (*s*) and albumin (gene symbol unknown). Cheng (1988, personal communication) found 36.9 percent recombination between yellow (*y*) and white-breasted (*wb*).

Tests that have revealed independent segregation between traits are summarized in Table 21.6.

Other poultry species. Genetic linkage information on the other avian species reviewed in this book is quite limited. This stems from several problems when compared to the chicken: longer generation interval, reduced numbers of progeny per mating, lower economic importance and therefore less funding available to support research, and less overall interest by hobbyists. Chicken hobbyists have obtained a great amount of

Table 21.5. Z-linked loci reported in Japanese quail.

Symbol	Trait name	Reference
al	Imperfect albino	Lauber (1964)
al^C	Cinnamon	Homma and Jinno (1969)
al^D	Dark-eyed dilute	Chahil and Johnson (1974)
al^{reb}	Red-eyed brown	Wakasugi and Kondo (1973)
br	Recessive brown	Homma (1968); Wakasugi and Kondo (1973)
ro	Roux	Somes (1988)

Table 21.6. Japanese quail loci showing independent segregation. References are listed at the end of the table.

Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref	Symbol	Tested against	Ref
Alb	*Es-D*	3	*Es-D*	*Alb*	3	*Pgi*	*W*	4	*Tf*	*Pgi*	3
	Hb	3		*B*	3		*Y*	4		*s*	4
	Pgi	3		*Hb*	3					*W*	4
	Tf	3		*Pgi*	3	*rf*	*E*	2		*Y*	4
				s	4						
B	*Es-D*	3		*Tf*	3	*rt*	*E*	1	*W*	*Hb*	4
	Hb	3					*sb*	1		*Pgi*	4
	Tf	3	*Hb-1*	*Alb*	3		*Y*	1		*Tf*	4
	Bh	5		*B*	3						
Bh	*B*	5		*Bh*	3	*s*	*Es-D*	4	*wb*	*E*	1
	Hb	3		*E*	3		*Hb*	4			
	Pgi	3		*Es-D*	3		*Pgi*	4	*we*	*Bh*	5
	Tf	3		*Pgi*	3		*Tf*	4			
	we	5		*s*	4				*wh*	*Bh*	5
	wh	5		*Tf*	3	*sb*	*E*	1		*Y*	1
	Y	5		*W*	4		*rt*	1			
E (e^{rh})	*Hb*	3		*Y*	4		*Y*	1	*Y*	*Bh*	5
	rf	2								*E*; e^{rh}	1
	rt	1	*Pgi*	*Alb*	3	*Tf*	*Alb*	3		*Hb*	4
	sb	1		*Bh*	3		*B*	3		*Pgi*	4
	Tf	3		*Es-D*	3		*Bh*	3		*rt*	1
	wb	1		*Hb*	3		*E*	3		*sb*	1
	Y	1		*s*	4		*Es-D*	3		*Tf*	4
				Tf	3		*Hb*	3		*wh*	1

References: 1-Cheng, 1988, personal communication; 2-mutation previously unreported, Cheng, 1988, personal communication; 3-Ito et al., 1988a; 4-Ito et al., 1988b; 5-Minezawa and Wakasugi, 1977.

information, developed purebred stocks that make excellent tester lines, and have stimulated interest in young people who later became researchers.

The Z-linked traits that have been reported in some species are listed in Table 21.7. The limited linkage map of the turkey Z chromosome is shown in Figure 21.4.

Only one autosomal linkage has been reported in the turkey, between glaucoma (*ga*) and slate (*D*) (Smyth et al., 1982). No recombination value was reported.

Lancaster (1963) reported no recombinants in the duck when autosomal black (*E*) and dominant bib (*S*) loci were tested, indicating allelism or very close linkage.

LINKAGE BETWEEN MORPHOLOGICAL AND MOLECULAR MARKERS

Other than the blood groups, few linkage studies between classical morphological markers and biochemical and molecular markers have been conducted. A number of molecular markers are available, such as restriction fragment length polymorphisms and loci mapped by *in situ* hybridization, but these have been underutilized in linkage studies. For example, six *ev* loci have been assigned to chromosome 1; the linear relationship

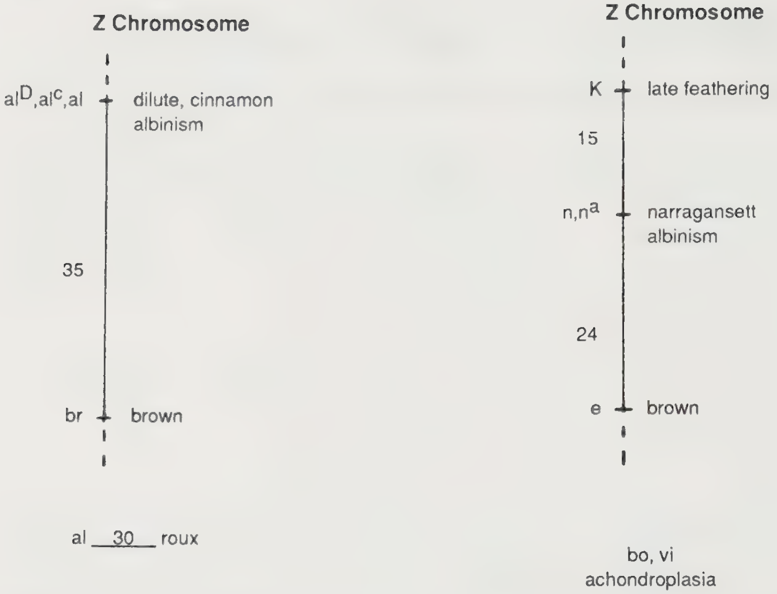

Figure 21.4. Map of the Z chromosomes of the Japanese quail (left) and the turkey (right).

Table 21.7. Z-linked loci reported in several economically important species of poultry.[1]

Species	Symbol	Trait name	Reference
Turkey	*ach*[2]	Achondroplasia	Abbott et al. (1965)
	bo	Bobber	Harper et al. (1988)
	e	Brown	Asmundson (1950)
	K	Late feathering	Asmundson and Abbott (1961)
	n	Narragansett	Robertson (1929)
	n^a	Albino	Hutt and Mueller (1942)
	vi	Vibrator	Coleman et al. (1960)
Ring-necked pheasant	*a*	Incomplete albinism	Bruckner (1969)
	Ba^+	Barring	Bhatnagar et al. (1972)
	di	Dilute	Wentworth et al. (1967)
	id^+	Dermal melanin	Bhatnagar et al. (1972)
	s^+	Gold	Bhatnagar et al. (1972)
Guinea fowl	$Acon^B$	Cytoplasmic aconitase	Baverstock et al. (1982)
	k	Sex-linked feathering	Micek and Malik (1970)
Domestic duck	*d*	Brown dilution	Punnett (1930, 1932)
	bu	Buff dilution	Lancaster (1963)
Muscovy duck	*ch*	Chocolate	Hollander (1970)
Goose	*G*	Grey[3]	Jerome (1970)
	Sd	Dilution[3]	Jerome (1953)
	Sp	Solid pattern	Jerome (1954, 1970)

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[1] Refer to appropriate chapter for gene description.
[2] Proposed gene symbol.
[3] Linked by two map units (Jerome, 1970).

between these loci is known, but they have not been tested against any morphological trait or chromosome rearrangement break point on this chromosome. Often molecular investigators have an interest in a particular locus or process, and are not equipped or motivated toward mapping work. The classical geneticist does not have the laboratory facilities or expertise to conduct the detailed procedures needed for molecular analysis.

Molecular studies could be utilized in conjunction with planned matings conducted to test other linkage relationships, and results analyzed as in classical linkage tests. Studies such as this would be extremely valuable in advancing our knowledge of the genetic map.

A number of chicken genes have been cloned. It should be a relatively short next step to utilize probes for these genes to study chromosomal locations. Testing probes against break points of chromosome translocations would be useful. There are about 20 translocation lines available, each with two break points. Some of these rearrangements involve microchromosomes.

Somatic cell hybridization could be conducted using chromosome translocations. If a translocation product is left after chromosome elimination, this would identify the region of a particular chromosome in which the locus in question is located. Hutchison (1987) suggested that the microchromosomes may be usable for gene mapping studies by pulse-field electrophoresis, creating another tool for use in defining genetic maps of birds.

The interests of investigators in various laboratories differ. It may be difficult to meld molecular and classical tests for a common goal, but it is a needed effort. It is relatively easy to obtain blood samples during the conduct of testcrosses that can be made available to other laboratories for molecular analysis. Results can then be analyzed to determine if any linkage relationships are apparent.

In applied breeding, this information may be of use in marker-assisted selection, where selection for an easily identified trait will improve a closely linked trait. Identification of molecular markers in inbred lines and in selection lines being maintained by breeders could be useful for identification of associations with productivity traits.

LINKAGE ANALYSIS

Green (1981) discussed systems used for testcrosses. The most basic, and perhaps the easiest is a reciprocal backcross. This requires the least number of progeny and reveals any sex differences in recombination rates. The simplest system to analyze is a double backcross, mating doubly heterozygous males to females recessive for the traits being tested. Once linkage is detected and initial estimates of distances are obtained, matings less efficient but more informative can be conducted.

Intercross matings can be inefficient. These may be the only possible matings, as when testing recessive embryonic lethals. More progeny are required, and it is not possible to follow recombination events in specific parents. The numbers in the double recessive class will be low, and if close linkage exists, the two single homozygous recombinant classes will also contain low numbers. Estimates of recombination frequencies are not reliable, because a difference of one observation can have a large effect. The double recessive may not even be recovered, skewing the recombination data. A double intercross of two codominant loci with fully classified progeny yields the largest amount of information (Robinson, 1971) but this cross is not always feasible.

The chi-square test is the standard statistical test conducted to determine if linkage exists between two traits. The null hypothesis is that the loci in question are not linked. As an example, a backcross with two dominant traits in coupling should have four classes of progeny, with equal numbers in each class. If the observed numbers deviate from the expected numbers by too large an amount, then the traits being tested are not segregating in an independent manner.

There are three tests required in analyzing a two-point testcross. Two of these analyses test segregation of each of the two traits to ensure that segregation is as expected. A distortion event may occur to transmit one allele in a higher frequency than the other allele. Segregation distortion has been reported in some species, but there have been no confirmed reports of this phenomenon in avian species.

Another difficulty might be incomplete penetrance, common with several traits in the chicken. The effect of the gene on segregation and viability must be known, and knowledge of interactions with other loci is also required. Testing in different genetic backgrounds can help clarify the expression of the gene. Once these factors are known, necessary corrections can be made during the analysis (see Robinson, 1971, for a more detailed discussion).

The third analysis examines the segregation of the two traits being tested to determine if they are being transmitted to the offspring in an independent fashion. The values obtained are compared to standard tables of probability to determine if there is a significant difference from the expected values. If a significant difference exists, then a recombination value (centimorgan = map unit value) with its standard error can be calculated.

Maximum likelihood estimation appears to be the method of choice for calculation of recombination values from planned test matings. Several detailed descriptions of this procedure have been published. Green (1963) presents a discussion and several worked examples drawn from mouse data, and includes a series of tables for use in refining estimates. Robinson (1971) presents a more detailed explanation of some of the theory, but does not include the tables that Green includes. Green (1981) presents a detailed discussion of the statistical theory and discusses various mating systems and associated analyses; tables similar to those presented by Green (1963) are not included.

Green (1963) is probably the best working paper of the three to follow for analyzing data. The other two papers are useful for background information and more detailed explanation of the theory used with maximum likelihood estimates. These are recommended readings for those conducting and analyzing linkage studies.

Results from several tests can be consolidated if authors present sufficient information in their reports. Parental genotypes, mating type (backcross, etc.), whether the traits were in coupling or repulsion, and the number of progeny in each class with the parental gametic types indicated, should be the minimum information presented. If only the total parental and total recombinant types are presented, not enough information is available to allow combining with data from other types of matings. With this information, later investigators working with the same loci can conduct an analysis for heterogeneity of data. If homogeneity exists the different studies can be combined. Information based on larger numbers with lower standard errors can be obtained. Green (1963) has an example of this procedure.

Claims of long linkage distances should be made with caution. Large numbers of progeny are needed to prove distances of around 35 map units or longer. Statistically it can be shown that approximately 600 individuals are needed to prove linkage of 45 map units at the five percent level of probability. There have been cases of possible spurious linkages being reported, where examination of larger numbers of progeny might have alleviated some confusion. Using a probability of .01 to claim linkage, rather than .05 might be useful if there are questionable results. Presentation of numbers in each class will also allow readers to use their judgement as to significance of results, considering some of the problems with penetrance and expression of many avian genes.

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Part III. NEW DIRECTIONS IN POULTRY GENETICS

Chapter 22. IMMUNOGENETICS

S.J. Lamont and R.R. Dietert

THE IMMUNE SYSTEM

General considerations. Given the importance of growth and overall productivity of poultry to the agricultural sector, the maintenance of health in these birds represents a significant factor in food-producing efficiency. The poultry immune system plays an active role in health maintenance; both genetic and environmental factors determine the success of this role in any given bird. Genetic factors tend to determine the ability of the bird to attack a particular disease organism, although selection for certain generalized immune parameters has also been achieved (Ubossi et al., 1985). Environmental factors (e.g. diet, stress, infections) tend to influence the efficiency of the immune response to a variety of challenges. Therefore, attempts to select for improved immune resistance to diseases must be tempered both by the environmental circumstances under which birds are evaluated for selection and the conditions under which selected birds are subsequently maintained for performance. In other words, the best genetic potential for disease resistance could be undone by an environment leading to suboptimal immune performance.

This chapter will concentrate on two major aspects of poultry immunogenetics: the identification and characterization of genes and gene products important for immune function, and the use of immune reagents for the analysis of blood cell markers. Topics related to poultry immunogenetics such as immune-based disease resistance and the relationship of oncogenes to immune cell tumorigenesis will be described in other chapters of this publication.

To fully consider the role of breeding and genetics in immune resistance to disease, it is necessary to examine the numerous possibilities for genetic modulation of the poultry immune system. The complexities of immune response to pathogenic challenge offer a variety of opportunities for genetic manipulation of immune surveillance either via breeding alone or through genetic engineering combined with selective breeding.

The immune system can be divided into two major branches, that giving rise to humoral immunity or antibody production and that giving rise to cell-mediated immunity or direct cellular intervention against pathogens. Humoral and cell-mediated immunity combine to provide birds with a complete spectrum of resistance processes. Each process is partitioned to function either in regional immunity (e.g. mucosal surfaces, skin, eyes) or as one step in a multipronged attack against a pathogen. An important feature of the immune response is the necessity of cell communication and cooperation. This occurs either by direct cell-cell contact between immune cells or by the interaction of secreted factors (cytokines) with cell surface receptors on responding cells. Because most immune effector functions are dependent upon multiple immune cell interactions, genetic influences are possible at several stages in the process.

The primary cellular constituents of the immune system are the bursa-derived (B) and thymus-derived (T) lymphocytes, and the inflammatory leukocytes (macrophages and granulocytes). It is the interaction between macrophages, T-, and B-lymphocytes that gives rise to immune-mediated protection from disease.

Appropriate differentiation of these immune cells is required for immune competency. As a result, genetic influences on the lymphoid organs that provide the microenvironments for immune differentiation and/or repertoire expansion could be expected to also influence immune function. Such examples are prominent in mammals with the genetically athymic nude (*nu/nu*) and hairless (*hr/hr*) mice lacking appropriate T-lymphocyte functions (Schultz and Sidman, 1987). In chickens, strains deficient in certain immunoglobulin classes (agammaglobulinemia) have been developed and studied (Benedict et al., 1977).

The immune system operates with a delicate balance between progressive and regressive signals in a given response. This help vs. suppression balance is also subject to genetic influences. In mammals, numerous cases of hereditary autoimmunity exist. Genetically based alteration of the immune and possibly other systems results in aberrant responses directed against self antigens. The New Zealand black mouse strain with a systemic lupus-like disease (Howie and Helyer, 1968) and the obese chicken strain with spontaneous autoimmune thyroiditis (Wick et al., 1981) are two prominent examples.

Finally, development of the capacity to fight infection is an important genetic consideration in poultry. The newly hatched chicken is provided with passive protection by maternal antibodies acquired via the egg. As these antibodies are lost through normal catabolism, the immune system of the chicken must begin to function both promptly and efficiently to meet the onslaught of pathogenic challenges occurring in the first few weeks after hatching. Recent evidence suggests that the early development of host immunocompetency is subject to genetic influences (Pitcovski et al., 1987a, 1987b). This is particularly important since the immune system of the neonatal chicken must win the race against infection to avoid reduced productivity and/or increased mortality.

Special poultry immune considerations. The immune system of poultry species has several important features distinguishing it from the mammalian immune system. These include differences in blood cell components (Chang and Hamilton, 1979), antibody molecule structures (Benedict and Berestecky, 1987), antibody gene family organization (Reynaud et al., 1987b), the lymphatic circulatory system (Toivanen et al., 1981), and the very nature of the inflammatory response (Nair, 1973; Grecchi et al., 1980). Of particular prominence is the spatial separation of avian B- and T-lymphocyte differentiation. The bursa of Fabricius located in the cloacal region of birds provides the microenvironment necessary for repertoire expansion of differentiating antibody-producing cells. There is no mammalian organ known to serve as the counterpart to the bursa. The proximity of the bursa to the vent also means that although the organ represents a central or primary lymphoid organ, it is readily exposed to external environmental factors via the sucking action of the vent (Sorvari et al., 1975). In this particular feature, it is more analogous to certain peripheral or secondary lymphoid organs. It has also been suggested that the chicken thymus, unlike that of mammals, has characteristics of a peripheral lymphoid organ by virtue of the presence of a sizable resident B cell population after hatching (Albini and Wick, 1974).

The physical separation of differentiating B- and T-lymphocytes in birds provides special considerations for the geneticist. Because the environments for differentiation of each lymphoid population are largely distinct, the opportunity exists for genetic modification of B- vs. T-microenvironments where the basis for selection-induced changes can be readily assessed. Thus far, this targeted immune selection approach involving the unique anatomical features of birds remains largely untapped.

GENE-ANTIGEN RELATIONSHIPS

The recent application of biochemical techniques to poultry immunogenetic analyses has been a powerful force in the identification of genetic heterogeneity within poultry flocks (Miller et al., 1984; Soller and Beckman, 1986). At the same time, the ability to characterize individual birds at the levels of serological specificity, protein structure, mRNA species and DNA structure has added complexity to the definition of an 'allele' and revealed the degenerate nature of the gene-antigen relationship.

Historically, the immunogeneticist identified allelic differences in birds based upon serological differences in blood cells (Landsteiner and Miller, 1924; Briles et al., 1950). While this procedure has been a potent method of genetic analysis, antigenic determinants are sometimes far removed from the underlying structural genes that the immunogeneticist is actually studying. The human ABO blood group is a prime example of the lack of a direct correspondence between genes and antigens. Because the A and B antigens of this system are determined by carbohydrate residues, they result from the post-translational modification of gene products. Therefore, the immunogeneticist identifying the A antigen in human populations is actually detecting the inheritance of a particular enzyme rather than the actual A antigen glycoprotein that the biochemist might isolate. In this case, the gene encoding the A enzyme and the antigen-containing gene product (the substrate) are not directly related. A mutant allele in either the A enzyme gene or the substrate gene might result in the lack of A glycoprotein production. Because carbohydrate antigens including the A antigen can sometimes occur on glycolipids in addition to glycoproteins, it is quite possible to have an antigen where the substrate cannot be linked to even one or two structural genes. As a result, the actual antigen used as a marker by the immunogeneticist can sometimes be the composite product of numerous structural genes. In contrast, there are several examples where expression of protein antigens is directly linked to inheritance of alleles of one or two structural genes. The relationship between expression of class I and II antigens and inheritance of alleles of corresponding *B*-complex genes would fall into this category (Crone and Simonsen, 1987).

Because the immunogeneticist can study immune-related inheritance at several levels of analysis, the concept of the 'allele' has become somewhat complex. Birds possessing the same antigenic determinant identified on the basis of serology and assumed to carry the same allele could actually have different alleles and antigen-containing molecules providing that amino acid substitution would not alter the antigenic determinant being studied. In this case, differences in structural genes and gene products would not be revealed by serology. Likewise, birds with identical protein gene products could have differences in mRNA and DNA sequences that would not result in biochemically discernable alleles at the protein level. Therefore, alleles can be defined at the level of nucleic acids, proteins or antigens. When poultry strains are now said to differ in alleles for a particular gene or to carry the same allele for a given gene, the level of the immunogenetic analysis is crucial in defining the actual relationship to DNA sequence comparisons.

IMMUNE REAGENTS

Immunological reagents have served as useful tools for the analysis of serological determinants associated with the products of particular poultry gene systems. Because of the specificity of the antigen-combining site of antibody molecules, antibodies have enabled researchers to quantitate levels of gene products and to distinguish between multiple alleles of certain polymorphic genes.

Polyclonal antisera. Originally, poultry immunogenetics depended almost exclusively upon alloantisera prepared by injecting cells from one animal into a recipient of the same species. If the animals differed in the inheritance of alleles for genes encoding cellular products and the recipients recognized the 'foreignness' of the gene products on donor cells, then the recipient would produce antibodies capable of reacting with allelic-associated portions of the donor cell proteins. Because the donor cells contained many proteins and each protein might be capable of eliciting more than one kind of antibody molecule, the response of the recipient was often directed against more than one antigenic determinant. In addition, the serum of the recipient bird would contain antibodies to antigens extraneous to the specific immunogen. As a result, the subsequent reagent was said to be polyclonal in that the antibodies it contained were the products of more than one B-lymphocyte.

Chicken polyclonal antisera grouped into series have been particularly important in the characterization of blood group alleles within poultry flocks. Much of the blood group analysis has been based on the hemagglutination assay. In this assay, antibodies with more than one binding site per molecule crosslink the antigen-bearing erythrocytes by overcoming the natural negative surface charge on the cells. Erythrocyte crosslinking can be scored visually by assessing the clumping of cells resulting from antibody reactivity.

While polyclonal antisera have the potential for detecting multiple antigens on cells and for having extraneous antibodies, they do offer some advantages as immunological reagents. Both mammalian and avian antisera will usually contain more than one class of immunoglobulin. Each class possesses a certain subset of effector functions (e.g. complement fixation, precipitation of antigen). Therefore, multiclass polyclonal antisera have the capabilities of performing most effector functions of antibodies. This means that such antisera can be used in a variety of immunological assays where agglutination, complement fixation, or immunoprecipitation may be desired. The utility of being able to apply the same polyclonal reagent to most assays is a major advantage for these reagents. One example of the adaptability of antiavian polyclonal antisera is the use of the same rabbit polyclonal antisera against chicken fetal antigen glycoproteins for such diverse assays as hemagglutination, complement-mediated cytotoxicity, immune electron microscopy, fluorescent microscopy, immunoprecipitation, and Western blotting analysis (reviewed in Dietert, 1987a, and in Sanders and Kline, 1988).

Monoclonal antibodies. Recently, products of hybridoma technology, monoclonal antibodies (Mabs), have become useful for analysis of avian gene products. These antibodies represent the products of single clonally expanded B-lymphocytes. As a result, the antibody preparations contain homogenous antibodies of limited antigen binding specificity. Since B-lymphocytes, at present, can not be grown in long-term cultures, immortalization of individual B-lymphocytes is an important feature of monoclonal antibody production. This is accomplished by the somatic hybridization of antigen-primed B-lymphocytes with myeloma (B-lymphocyte derived) tumor cells. When the parental myeloma cells are permissive for antibody production but do not themselves synthesize

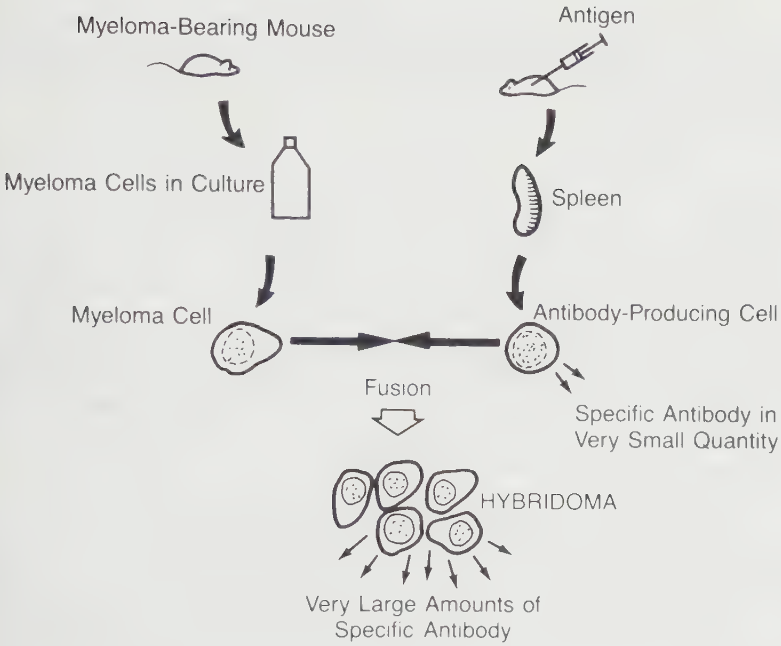

Figure 22.1. A generalized strategy for the production of monoclonal antibodies from a hybridoma. (From Lamont et al., 1987b).

immunoglobulin polypeptides, the hybrid cell can produce antibody derived from the donor B-lymphocyte immunoglobulin genes (Kohler and Milstein, 1975). Therefore, the B-lymphocyte contributes the particular antibody to the hybrid while the myeloma cell conveys the phenotype of enhanced proliferative capacity. A diagram describing the general preparation of mouse monoclonal antibodies is shown in Figure 22.1.

Because monoclonal antibodies are homogeneous, they exhibit exquisite specificity as immunological reagents. The ability to grow antibody-producing hybridomas either in culture or as ascites tumors *in vivo* provides the benefit of a continuing supply of the antibody. A limitation of monoclonal antibodies relates directly to the homogeneity of the reagents. Since only one class or subclass of immunoglobulin will normally be present in a Mab preparation, the antibody will perform different effector functions with different efficiencies. A given Mab might agglutinate and react with complement well but be inadequate in assays requiring immunoprecipitation. For this reason, it is advisable that Mabs of desired specificity be selected using screening assays that reflect the intended primary use of the Mab. One alternative to this approach is the recently described method of selecting variant Mab-producing clones that have undergone a desired immunoglobulin class switch (Waldor et al., 1987). A clone producing IgM Mab could be driven to produce an IgG subclass (employing a downstream heavy chain gene). However, because the process of class-switching usually involves deletion of DNA material, an IgG-producing hybridoma could not easily be made to produce IgM. A second approach has been to genetically engineer antibodies to form chimeric molecules with the antigen-binding specificity derived from one antibody and the effector function from a second antibody (Liu et al., 1987).

Monoclonal antibodies to poultry gene products date back to the 1970s (Longenecker et al., 1979). Thus far, these antibodies have been produced against a variety of antigens with the greatest emphasis being placed on products associated with the hematopoietic system. Numerous examples of the application of Mabs to poultry immunogenetics are indicated throughout this chapter.

DEVELOPMENT AND USE OF SPECIALIZED STRAINS

Studies in avian immunogenetics are aided by using specialized reagents, such as polyclonal and monoclonal antibodies, and also by using specialized genetic strains of birds. Specialized strains can help answer questions about genetic control of diverse phenomena such as blood group antigens, differentiation antigens, lymphocyte antigens, immunoglobulin production, immune response, and disease resistance. Examples of different types of specialized genetic strains that have been used in studies of avian immunogenetics are inbred lines, congenic lines, aneuploid lines, and lines selected for single gene or polygenic control of immune response traits.

Inbred lines. Inbreeding and its effects are discussed in Chapter 39 and elsewhere (Altman and Katz, 1979; Hála, 1987; Abplanalp, 1986; Stone, 1975). The advantage of using inbred lines is that each line is genetically homogeneous but that genetic diversity can exist between lines. Inbred lines of chickens have been used extensively for immunogenetic studies throughout the world including laboratories in Australia (Sheldon, unpublished), Iowa (Rees and Nordskog, 1981; Gebriel and Nordskog, 1983), Czechoslovakia (Hasek et al., 1966), England (Payne and Chubb, 1968), California (Benedict et al., 1975), Michigan (Stone, 1975), Finland (Toivanen and Toivanen, 1973), and Switzerland/Denmark (Simonsen et al., 1982; Pink et al., 1985b).

MHC-congenic lines. Congenic lines are lines which differ from each other in only a small chromosomal segment containing one or a few genes, or in chickens perhaps differing in a microchromosome. Lines congenic for the MHC have been extremely useful in investigating the function of the chicken MHC. Congenic lines can be developed by three different methods (Hála, 1987). Homozygous sublines can be developed from the residual heterozygosity in an inbred line, or recombinant animals can be identified in an inbred line, or congenic lines can be produced by backcrossing the gene of interest into an inbred line for preferably ten generations (Figure 22.2). Lines congenic for the MHC have been developed and studied in Czechoslovakia (Hasek et al., 1966; Hála et al., 1966; Hála, 1987), Michigan (Shen et al., 1984), and California (Abplanalp, 1986) (Table 22.1). Additional congenic lines for the MHC are currently under development in New Hampshire on the UCD-003 background, in Illinois on an Ancona background, and in Iowa on the HN and G backgrounds.

Aneuploid birds. The identification by Bloom and co-workers (Bloom and Bacon, 1985; Bloom et al., 1987, 1988) of birds which were aneuploid for the MHC-bearing chromosome allowed the development of a trisomic line of chickens that has been extremely useful in mapping the MHC and studying MHC gene dosage effects, as described later in this chapter.

Selected lines. Selected strains, in which selection was based upon some measure of immune response, have proved to be valuable in immunogenetics studies. Lines have been

Table 22.1. MHC (*B*)-congenic chicken lines

		MHC		
Line	*B* haplotype	*B-L*	*B-F*	*B-G*
Prague MHC congenic lines on CB background[1]				
PR-CB	12	12	12	12
PR-CA	12	12	12	12
PR-CC	4	4	4	4
PR-CB.I-B7	7	7	7	7
PR-CB.R1	12r1	12	12	4
PR-CC.R1	4r1	4	4	12
PR-CC.R2	4r2	4	4	7
PR-CA.I-M-1	12	12	12	12
Prague MHC congenic lines on WA background[1]				
PR-WA	9	9	9	9
PR-WB	15	15	15	15

Line	*B* haplotype	*B* donor line
East Lansing MHC congenic lines on $15I_5$ background[1]		
RPRL-$15I_5$	15	Inbred base
RPRL-15.6-B^{2}	2	RPRL-6_1
RPRL-15.7-B^{2}	2	RPRL-7_2
RPRL-15.15I-B^{5}	5	RPRL-15.I_4
RPRL-15.C-B^{12}	12	RH-C
RPRL-15.P-B^{13}	13	JM-P
RPRL-15.P-B^{19}	19	JM-P
RPRL-15.N-B^{21}	21	JM-N
University of California-Davis MHC congenic lines on UCD-003 background[2,3]		
UCD-003	17	Inbred base
UCD-254	15 (15)	UCD-007
UCD-253	6	UCD-002
UCD-330	X (21)	UCD-100
UCD-331	Y (2)	Hy-Line
UCD-335	Z (19)	UCD-159
UCD-312	24 (24)	UCD-200
UCD-316	14	UCD-400
UCD-336	Q (21-like)	UCD-001
UCD-333	J	UCD-001
UCD-396	L	UCD-132
UCD-314	21 (21)	Hy-Line
UCD-313	3 (3)	UCD-071
UCD-104	0	UCD-500
UCD-342	C	UCD-500
UCD-?	15 (15)	Hy-Line

[1] Adapted from Hála, 1987.

[2] Adapted from Abplanalp, 1986.

[3] UCD *B* haplotype given first, Briles *B* haplotype given in parentheses if known.

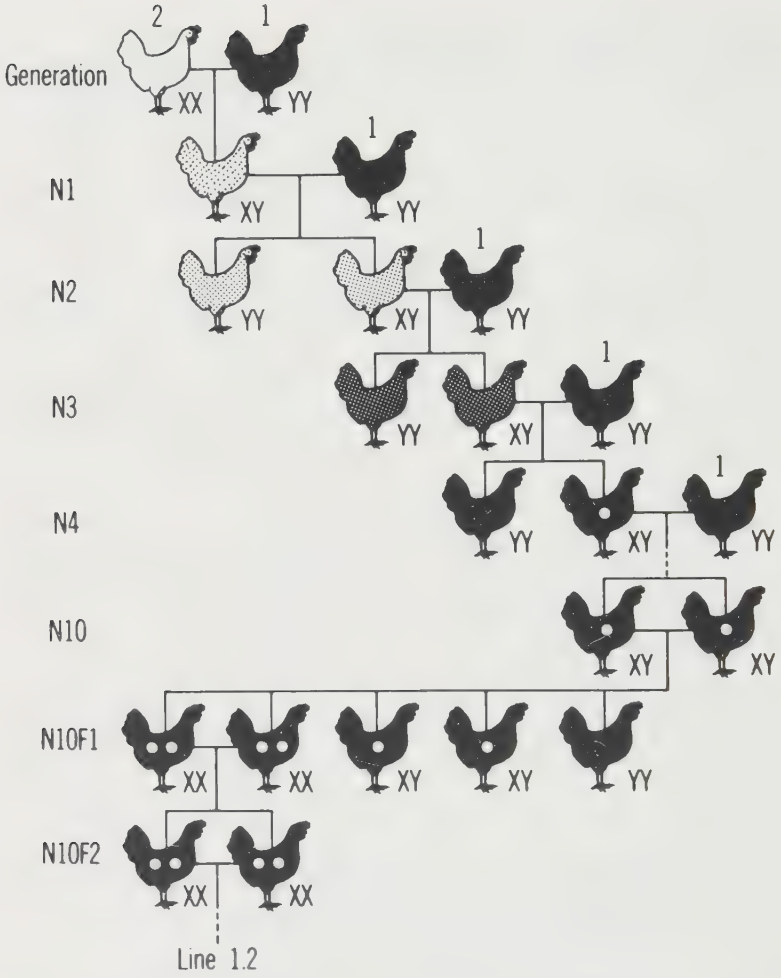

Figure 22.2. Production of a congenic line of chickens. Strain 2, which has the gene *X* of interest, is crossed with inbred strain 1. The F_1 hybrid is then backcrossed to strain 1, which carries the *Y* allele. Progeny are evaluated and heterozygous *XY* birds are again backcrossed to strain 1. After ten generations of backcross matings, full sibs bearing *XY* are intercrossed. The homozygous *XX* progeny of this mating are intercrossed to establish the Line 1.2, which is congenic with strain 1. In designating a congenic line, the background strain precedes the decimal point and the source of the gene of interest follows the decimal. (Adapted from Klein, 1982).

selected for a variety of immunological traits including level of antibody response to *Salmonella pullorum* antigen (Pevzner et al., 1981), and SRBC (Siegel and Gross, 1980), early antibody titres to *E. coli* and Newcastle disease virus vaccine (Pitcovski et al., 1987a, 1987b), and large and small bursa size lines (Yamamoto and Glick, 1982). The genetic control in many cases seems to be polygenic. The use of long-term selected lines allows evaluation of the genetic parameters of selection for immune response and correlated traits. Lines selected and inbred for disease resistance or susceptibility have been widely used in subsequent studies of immune function (see Chapter 33).

BLOOD GROUPS

Blood groups in chickens. Blood group antigens in chickens have been studied primarily by using alloantisera in a hemagglutination assay. The alloantisera are produced by immunization with erythrocytes between chickens differing at one or more blood group loci. The resultant antisera may contain antibodies against several antigens. It may be useful to make the antisera more specific by adsorptions with erythrocytes to remove particular antibodies from the solution (Gilmour, 1960; Briles et al., 1950). The removed antibodies will be the ones which recognize antigens on the adsorbing erythrocytes. To test for the presence of a blood group antigen on the red cells of an individual bird, a dilute sample of red cells is mixed with an appropriate dilution of a reagent alloantiserum. After an incubation period, the red cells will be crosslinked (agglutinated) if they bear antigens recognized by the antiserum. Because there are often cross reactions between different allelomorphs, it is useful to test red cells with a series of alloantisera for which the characteristic pattern of reaction with standard alleles is known. Identification of the sample blood type is then made by comparison with the reaction pattern of the series of reagents on cells of known allelic types.

The pioneering work on chicken blood group antigens was conducted by W.E. Briles in the U.S.A. beginning in the late 1940s and, independently by D.G. Gilmour in England. Prior to that time, however, Landsteiner had demonstrated that individual antigens on chicken erythrocytes could be recognized by adsorbed rabbit anti-chicken serum (reviewed in Landsteiner, 1945). Todd (1930) used alloantisera to show that chicken erythrocyte alloantigens are under genetic control.

The most immunogenic erythrocyte antigens, designated system *A* and system *B*, were the first to be characterized genetically and serologically (Briles et al., 1950). Each gene system was shown to be encoded by two autosomal genes with multiple alleles. The allelic products were demonstrated to be expressed in a codominant fashion, as was subsequently found to be the case for all other erythrocyte antigens. Two additional blood group systems, *C* and *D*, were identified next and were shown to segregate independently of each other and of the *A* and *B* systems (Briles et al., 1950, 1963; Briles, 1951, 1962). Next, a fifth blood group system *E* was detected and shown to be closely linked to the *A* system (Briles et al., 1959). Only about one percent crossingover occurs between *A* and *E*. Blood group loci and antigens can also be designated as *Ea-A*, *Ea-B*,... and Ea-A, Ea-B,..., respectively, to indicate that they represent erythrocyte alloantigens (Gilmour, 1985).

Gilmour was studying erythrocyte antigens in chickens concurrently and upon comparison of reagents produced by Gilmour and Briles, it became apparent that Gilmour had independently identified the *A*, *B*, and *C* blood group systems as well as two other systems, *L* and *N* (Gilmour, 1959). A common finding of both Briles et al. (1957) and Gilmour (1959) was the persistence of segregation for blood group loci in the presence of inbreeding. It was hypothesized that heterozygous individuals had an advantage in reproductive performance and survival which resulted in continued segregation for blood group loci (Gilmour, 1960, 1962).

Additional blood groups have been identified, bringing the total number up to 13: *A*, *B*, *C*, *D*, *E*, *H*, *I*, *J*, *K*, *L*, *N*, *P*, *R* (Briles, 1984). With the exception of the closely linked *A* and *E*, all other blood group loci showed independent segregation. All are autosomal.

The blood group antigens are identified primarily by their expression on erythrocytes, but some of the blood group antigens also are expressed on lymphocytes. Schierman and Nordskog (1962) demonstrated the presence of B and C antigens on white blood cells by using lymphoagglutination. Wong et al. (1972), using a novel allofixation technique, detected A and B antigens on lymphocytes.

The blood group antigens of different systems are not all detectable at adult levels at the same time point in ontogeny. For example, A antigens can be detected on blood cells from early (72-hour) embryos at a degree of agglutination which approximates that of adult cells (Briles, 1964). The B antigens, however, are not expressed at adult levels until 5-15 days posthatching.

The function of most of the chicken blood group systems is not known. The *B* blood group, however, serves as a marker for the major histocompatibility complex (Briles and Briles, 1987) as described later in this chapter. The *C* blood group is, or is closely linked to, a minor histocompatibility locus (Schierman and Nordskog, 1965). The *K* system is the genetic basis of the agglutinability of chicken erythrocytes with vaccinia virus (Brown et al., 1973). The *R* system has an allele associated with susceptibility to subgroup B leukosis sarcoma viruses (Crittenden et al., 1970). The R^1 antigen was isolated and shown to be similar to the human blood group N which also controls a viral receptor and the immunodominant structure of a blood group antigen (Springer et al., 1974). The significant alteration of blood group allelic frequencies concurrent with selection for immune response or disease resistance (Johnson and Edgar, 1984; Dunnington et al., 1984; Martin et al., 1986) suggests that some blood group systems may have important but as yet undiscovered functions.

Although specific functions are known for only a few of the chicken blood group systems, it is still useful to characterize blood groups in poultry populations. These loci show extensive polymorphism and are distributed throughout the genome. Thus, they can serve as markers to evaluate the genetic structure and purity of a population. Additionally, some loci as indicated have known functions and therefore it can be beneficial to selectively alter the frequency of certain blood group alleles in a population.

Blood groups in turkeys, Japanese quail, ring-necked pheasants, and ducks. Blood groups have not been as thoroughly investigated in any other avian species as they have been in the chicken. There have, however, been studies of blood groups in turkeys, Japanese quail, ring-necked pheasants and ducks (see Table 22.2). In turkeys, as is the case for chickens, there appear to be several blood group systems, some of which show strong evidence for multiple allelism (Briles, 1984; Law et al., 1968; Law, 1974). Little is known of the structure or function of turkey blood groups, but recent work suggests that blood groups in the turkey may also be linked with the major histocompatibility complex (Ellis et al., 1988).

In Japanese quail, three alloantigens have been identified on erythrocytes (Katoh and Wakasugi, 1980). These antigens were found to be controlled by three separate autosomal dominant genes, designated *Ea-QN*, *Ea-R* and *Ea-H*, and renamed *A*, *B*, and *C*, respectively (Katoh and Wakasugi, 1981). Two alloantigens, Ly_1 and Ly_2, have also been identified on erythrocytes, peripheral leukocytes, and splenocytes and found to be under autosomal codominant control (Katoh and Wakasugi, 1981). It was suggested that the *Ly* locus might be linked with *A* or $Ea\text{-}QN_1$, and that this locus might be associated with the major histocompatibility complex of Japanese quail.

Three different blood groups, designated *A*, *B*, and *C*, have been identified by using xenogeneic and allogeneic antisera on erythrocytes of ring-necked pheasants (Vohs, 1966). These antigens were inherited in a Mendelian fashion and segregated independently. One blood group system in ring-necked pheasants has been suggested to be associated with the major histocompatibility complex (Jarvi and Briles, 1988). The cells of individual

Table 22.2. Blood groups and agglutinogens in chickens, turkeys, and Japanese quail. (Adapted from Somes, 1988).

Species	Gene	Trait
Blood groups		
Chicken	*B-F*	MHC antigen B-F
	B-G (*Ea-B*)	MHC erythrocyte alloantigen B-G
	B-L	MHC leukocyte alloantigen B-L
	Ea-A	erythrocyte alloantigen A
	Ea-C	erythrocyte alloantigen C
	Ea-D	erythrocyte alloantigen D
	Ea-E	erythrocyte alloantigen E
	Ea-H	erythrocyte alloantigen H
	Ea-I	erythrocyte alloantigen I
	Ea-J	erythrocyte alloantigen J
	Ea-K	erythrocyte alloantigen K
	Ea-L	erythrocyte alloantigen L
	Ea-N	erythrocyte alloantigen N
	Ea-P	erythrocyte alloantigen P
	Ea-R	erythrocyte alloantigen R
Turkey	*Ea-A*	erythrocyte alloantigen A
	Ea-C	erythrocyte alloantigen C
	Ea-F	erythrocyte alloantigen F
	Ea-J	erythrocyte alloantigen J
	Ea-K	erythrocyte alloantigen K
	Ea-L	erythrocyte alloantigen L
	Ea-Q	erythrocyte alloantigen Q
Japanese quail	*Ea-H* (*C*)	erythrocyte alloantigen H (C)
	$Ea\text{-}QN_1$ (*Ly* or *A*)	erythrocyte alloantigen QN (A)
	Ea-R (*B*)	erythrocyte alloantigen R (B)
Agglutinogens		
Chicken	At, at^+	*Allium tuberosum* agglutinogen
	$Gs\text{-}1$, $gs\text{-}1^+$	soybean agglutinogen
	Hi, hi^+	*Lathyrus cicera* agglutinogen
	$Ln\text{-}1$, $ln\text{-}1^+$	*Lepista nuda* agglutinogen
	Mp_1, mp_1^+	*Maclura pomifera* agglutinogen 1
	Mp_2, mp_2^+	*Maclura pomifera* agglutinogen 2
	Ph, ph^+	garden pea agglutinogen
	$Pn\text{-}1$, $pn\text{-}1^+$	peanut agglutinogen
	Pp, pp^+	'Pp' agglutinogen
	Pw_1, pw_1^+	pokeweed agglutinogen 1
	Pw_2, pw_2^+	pokeweed agglutinogen 2
	Si, si^+	sesame agglutinogen
	Ssb, ssb^+	soybean agglutinogen
	St_1, st_1^+	potato agglutinogen 1
	St_2, st_2^+	potato agglutinogen 2
	Tg_1, tg_1^+	tulip agglutinogen 1
	Tg_2, tg_2^+	tulip agglutinogen 2

Continued...

Table 22.2. Blood groups and agglutinogens in chickens, turkeys, and Japanese quail. (Adapted from Somes, 1988). (Continued)

Species	Gene	Trait
	Va, va$^+$	*Viscum album* agglutinogen
Japanese quail	*Ht, ht*$^+$	*Helianthus tuberosus* agglutinogen
	Ns, ns$^+$	*Naematoloma sublateritium* agglutinogen
	Pn, pn$^+$	peanut agglutinogen
	Ps, ps$^+$	*Pisum sativum* agglutinogen
	Sb-1, sb-1$^+$	soybean agglutinogen
	Sn, sn$^+$	soybean agglutinogen

pheasants demonstrated differential reactivity with chicken reagents against *B-G*, *B-F*, and *Ea-A*, suggesting that equivalent systems exist in pheasants.

Studies on the blood group antigens of ducks were conducted in the 1930s and 1940s at the University of Wisconsin (reviewed in Briles, 1964). Matings of muscovy ducks with either Pekin or mallard ducks produced hybrids that had unique 'hybrid' substances, or red cell antigens distinct from those of either parent (McGibbon, 1944). Six or seven blood group loci seemed to be present in each of muscovy, Pekin, and mallard ducks. The A1-A2 antigens are controlled by an extensive series of multiple alleles (McGibbon, 1945). One of the mallard antigens was found to be sex-linked (McGibbon, 1946).

Agglutinogens. Agglutinogens on red cells are surface molecules, particularly glycoproteins, which are specifically bound by plant lectins. The multiple binding sites of the plant proteins can crosslink the erythrocytes and cause agglutination. The lectins bind specifically to sugars and oligosaccharides on the cell surface. The presence of many agglutinogens is under known genetic control (Table 22.2).

DIFFERENTIATION MARKERS OF BLOOD CELLS

The expression of specific genes during hematopoietic cell differentiation has proven relevant to the particular functional capacities of the cells. A primary example of this direct relationship is the differential expression of the CD4 and CD8 gene products on subpopulations of human T-lymphocytes. The presence of these proteins on the cell surface of T-lymphocytes appears to be significant in determining the particular immunoregulatory function of the cell (e.g. helper vs. suppressor function) (Reinherz and Schlossman, 1980). Most cell surface differentiation markers have been detected with the use of specific antibodies. Like their mammalian counterparts, avian blood differentiation markers have provided a means of identifying cell population heterogeneity within a lineage and dissecting the relationship between gene expression, protein structure, and cell function. Table 22.3 lists several differentiation markers of avian blood cells by cell type categories. A variety of immunological reagents has been developed to permit detection and/or quantitation of these gene products on the cell surface. These include avian alloantisera, mammalian polyclonal antisera frequently absorbed for specificity, and mammalian monoclonal antibodies. Given the utility of the latter category of reagents for analysis of

Table 22.3. Avian differentiation markers by cell types.

Predominant cell type category	Identifying antibody/antigen or differentiation marker
Hematopoietic cells	B-F (Class I); B-G (Class IV); Transferrin receptor (Tfr); chicken fetal antigen (CFA); chicken adult antigen (CAA); quail fetal antigen (QFA); CA1
Leukocytes	B-L (Class II); Fc receptor (FcR); CLA
T-lymphocytes	Th-1; Ly-4; TA; AT-1.1; CD4; CD8; T3; $T_{10}A_6$
B-lymphocytes	Ig; Bu-1; CB-1-CB-5

cell surface molecules, it is likely that the list of avian differentiation markers will grow rapidly in the next few years.

Detection of differentiation molecules. Differentiation molecules have been detected with antibodies using a variety of assays with several applications. For erythroid-associated markers, hemagglutination or crosslinking of cells by antibodies has commonly been employed. In contrast, immunofluorescence has been the most widely utilized assay for nonerythroid blood cells. In particular, indirect immunofluorescence using a secondary antibody labelled with a fluorochrome has been useful on both tissue sections and free blood cell preparations. More recently, enzyme-linked antibodies have permitted immunocytochemical analysis of blood cell markers. One advantage of this procedure is that more cell structure and architecture can be seen with the light microscope enzyme-dependent analysis than is usually visible under the dark field conditions of immunofluorescence. When quantitative analyses are required, the spectrophotometrically-read enzyme-linked immunoadsorbant assay (ELISA) and flow cytometry quantitation of immunofluorescence are both desirable alternatives.

The detection of antibody-antigen binding using radioactive labelling of either antibody or cellular antigen has facilitated the opportunity to perform biochemical analyses of blood cell differentiation markers. Use of antibodies to either identify the antigenic molecule or to isolate large quantities of antigen via an immunoadsorbant matrix has perhaps been the most powerful use of antibodies in the analysis of differentiation gene products.

Erythrocytes. Gene products associated with the differentiation and development of poultry erythrocytes have been described by several laboratories. Most of these products appear to be monomorphic, based on the apparent lack of segregation of antigens in different strains. The exceptions to this generalization are the major histocompatibility (*B*) complex associated B-F (class I) and B-G gene products expressed on erythrocyte cell surfaces. Both B-F and B-G products are highly polymorphic and have been detected using both alloantisera (Ziegler and Pink, 1976) and mouse monoclonal antibodies (Longenecker et al., 1979; Pink et al., 1985a; Crone et al., 1985). B-G proteins have been shown to be expressed primarily on erythrocytes in a pattern associated with erythroid differentiation and development. As such, B-G proteins comprise one of several cell surface gene products that appear on an increasing incidence of peripheral erythrocytes during neonatal development and have been termed chicken adult antigens (CAA) (Nelson et al., 1982). A second unrelated group of cell surface proteins, initially detected on chicken erythrocytes with decreasing incidence during neonatal development, has been termed chicken fetal antigens (CFA). CAA and CFA represent multiprotein serologically-complex antigen systems initially detected with polyclonal antisera. Individual protein components were later identified with monoclonal antibodies (Sanders et al., 1982; Miller et al., 1982). While protein components of the CAA and CFA system are under developmental control linked to erythroid differentiation patterns, at least some of the epitopes appear to involve carbohydrate moieties (Lewin and Dietert, 1983). A parallel fetal antigen system has been described in Japanese quail (Qureshi et al., 1983) that has molecular components related to CFA (Dietert et al., 1984). Some of the CAA and CFA proteins and associated antigenic determinants have been detected on nonerythroid hematopoietic cells as well (Dietert et al., 1982, 1983; Kline et al., 1988a). Little is known about the possible function(s) associated with CFA and CAA glycoproteins.

The transferrin receptor represents a cell surface component found on many vertebrate cells. The receptor functions to facilitate the incorporation of iron into cells via the transferrin carrier protein. Elevated expression of the transferrin receptor has been associated with cell proliferation (Neckers, 1984) and with slowly proliferating or nonproliferating cells having a high iron requirement such as erythrocytes (Iacopetta et al., 1982).

Mouse monoclonal antibodies were used to identify and characterize the chicken transferrin receptor (Schmidt et al., 1985). Proteins of 90,000 and 95,000 daltons molecular weight were isolated from avian erythroblastosis virus-transformed chicken erythroblasts using the JS8 monoclonal antibody. The 90,000 dalton protein appears to be a precursor of the larger molecular weight product (Schmidt et al., 1985). While the JS8 antibody did not block binding of transferrin to the receptor, it did inhibit iron uptake and erythroid differentiation by preventing receptor recycling (Schmidt et al., 1986b). The transferrin receptor has been used for analysis of chicken erythroid differentiation and to examine transformed macrophages (Schmidt et al., 1986a).

Leukocytes. A family of cell surface glycoproteins has been found to occur in common on the leukocytes of both mammalian and avian species. This family has been termed either the 'common leukocyte antigen' (CLA) or the 'T-200 glycoproteins'. Although the exact function of the molecules is unknown to date, the occurrence of the molecules on leukocytes but not mature erythrocytes has led to the suggestion that they may play a role in migration and/or cell differentiation. This is further supported by the observation that different leukocyte lineages (e.g. lymphocytes, macrophages, granulocytes) exhibit different forms of the T-200 molecules. Lineage-specific glycoprotein differences occur in the N terminus end of the molecules and may originate from alternate splicing of mRNA (Barclay et al., 1987). Quantitative differences in T-200 expression have also been reported during the maturation of specific human leukocyte lineages (Shah et al., 1988).

Monoclonal antibodies that appear to detect chicken CLA molecules include L.17 (Pink and Rijnbeek, 1983) and JS6 (Schmidt et al., 1986a). An antigen termed CLA-1 has been identified by a monoclonal antibody of the same name. This antigen is detectable on largely mononuclear leukocytes and is retained by T-lymphocytes during development (Chen et al., 1984). The CLA-1 antigen was detected in the chicken as two noncovalently linked polypeptide chains of Mr 180,000 and 195,000.

Lymphocytes. Several antigens have been detected that are associated with the differentiation of B- or T-lymphocytes. Most of these antigens were detected with the use of alloantisera although in some cases equivalent monoclonal antibodies have recently been prepared against the same antigens.

For B-lymphocytes, several alloantigen systems have been characterized. The Bu-1 alloantigens were initially identified in RPRL6_3 and RPRL7_2 chicken strains carrying the same B^2 haplotype and studied using both complement-mediated cytotoxicity and immunofluorescence assays (Gilmour et al., 1976; Brand et al., 1983). Alloantisera prepared by reciprocal immunizations between the lines demonstrated the existence of two Bu-1 alleles (Gilmour et al., 1976). Bu-1 antigen is inducible on B-lymphocytes by bursin and the incidence of the antigen on B-lymphocytes increases during embryonic development reaching approximately 85 percent at hatching (Brand et al., 1983). Cells capable of being induced to express Bu-1 have been found in the bone marrow and the bursa of Fabricius but are absent from the thymus (Brand et al., 1983). While the linkage association of the Bu-1 gene is unknown, it is not linked to the B-complex or to the Th-1 and Ly-4 T-lymphocyte alloantigen systems (Lehtonen et al., 1987). Recently, Bu-1 antigens have been studied using mouse monoclonal antibodies in place of chicken alloantisera (Pink and Rijnbeek, 1983).

The BA alloantigen was identified by alloantisera where bursal cells were used as immunogens and adsorption was conducted with thymus cells. This alloantigen appears to be present on virtually all bursal lymphocytes and approximately one-half of peripheral B-cells (Galton and Ivanyi, 1977).

Several monoclonal antibodies have been developed that react with antigens found primarily on B-lymphocytes. The antibodies and corresponding antigens have been designated as CB-1 to CB-5. Reactivity of CB-3 to CB-5 appeared to be restricted to B-lymphocytes (Chen and Cooper, 1987). Some biochemical characterization of the antigens has been performed and this panel of antibodies would appear to offer tools for dissecting the process of B-cell differentiation. At present, the nature of the genes encoding these antigens and the function of the gene products have not been determined.

Immunoglobulin (Ig) molecules serve as cell surface receptors of B-lymphocytes. Because these proteins are expressed during the commitment of lymphoid cells to the B-cell lineage, these gene products also serve as differentiation markers. The existence of multiple heavy chain class immunoglobulin genes which are expressed in a specific sequence further adds to the usefulness of Ig cell surface molecules as indicators of the extent of B-cell differentiation. This topic is considered in more detail later in this chapter.

Class II glycoproteins encoded by the B-L region of the chicken major histocompatibility (*B*) complex represent a cell surface differentiation marker associated with B-lymphocytes (Ewert and Cooper, 1978). These heterodimers (Mr 32,000 and 27,000) function in interleukocyte communication and are also expressed by certain T-lymphocytes (Calnek et al., 1984) and macrophages (Peck et al., 1982). A detailed consideration of these differentiation gene products is presented later in this chapter.

Several T-lymphocyte differentiation makers have been described in the chicken. Those detected with alloantisera where segregating alleles were involved include the Th-1 (Fredericksen and Gilmour, 1981), Ly-4 (Fredericksen et al., 1977), TA (Galton and Ivanyi, 1977), and AT1.1 (Wayes et al., 1984) systems. Of these markers, TA appears to be present on most T-lymphocytes regardless of location. AT1.1 and Th-1 are expressed by a majority of thymocytes with the latter present on a subpopulation of circulating T-lymphocytes (Lehtonen et al., 1987). In contrast, Ly-4 is expressed by most peripheral T cells but only a subpopulation of thymocytes (Fredericksen and Gilmour, 1981).

Recently described monoclonal antibodies specific for T-lymphocyte cell surface molecules appear to partition cell populations based on their level of differentiation. For example, the CT-1 antibody is predominantly thymocyte-specific in its reactivity. An antibody produced by Houssaint et al. (1985) designated as $T_{10}A_6$ reacts with thymocytes but also a subpopulation of peripheral T-lymphocytes. In contrast, the CT-3 Mab reacts with both thymocytes and most peripheral T-cells probably reacting with the T cell receptor (T3/Ti) complex (Chen et al., 1986). In contrast, the LD16 Mab appears to be specific for a T cell subpopulation that includes Ia-positive T-cells (Hála et al., 1985). Reactivities of two other anti-T-cell Mabs, 11A9 and 3G6, were reported to differ both for thymocyte and peripheral T-cell populations (Vainio et al., 1987) and two additional Mabs were recently used to identify the avian homologues of mammalian CD4 and CD8 T-cell subpopulation antigens (Chan et al., 1988).

As the number of immune reagents specific for functionally significant cell surface molecules increases, the ability of the poultry breeder to assess the impact of genetic modifications on poultry hematopoietic cell populations will be greatly enhanced. The importance of such opportunities to the geneticist is to permit a mechanistic evaluation of the impact of selection influencing immune function.

IMMUNOGLOBULIN GENETICS

Immunoglobulins (Igs) or antibody molecules represent the antigen-specific receptor of B-lymphocytes. As such, they play a pivotal role in humoral immune protection. The breadth of the protection is dependent in part on the diversity of Ig specificities for antigens. The specificity of the humoral immune response results from the fact that each B-lymphocyte displays an Ig cell surface receptor with identical or similar antigen binding specificities to the Igs that will be secreted upon B-cell stimulation. Therefore, only B-lymphocytes with appropriate surface Igs will be triggered to respond to a given pathogen.

The challenge to the animal is to produce the approximately one million different Ig specificities necessary for the full spectrum of a humoral protection without occupying a majority of the genome with Ig genes. While this genetic problem has been overcome in similar ways by both avians and mammals, there are some important distinctions in the genetic strategies utilized by these two groups to maximize Ig diversity. Strategies for achieving Ig diversity will be described later in this section following a description of Ig protein heterogeneity.

Ig structure. The Ig molecule appears to have evolved from a base unit structure of approximately 110 amino acids in length. This unit is termed a domain and contains an internal disulfide bond resulting in a loop of approximately 60 amino acids. The closest successor to the primordial Ig genetic structure is probably represented by the ß-2 microglobulin protein. This molecule closely resembles a single Ig domain. Ig polypeptide chains are composed of several repeated domains each having evolved a particular functional significance for the entire molecule. The NH_2 end domain of each chain contains the antigen-binding region and because of the great diversity of amino acid sequences in different Ig chains is referred to as the variable (V) region. The COOH end of the chain is relatively invariant and has thus been termed the constant (C) region. As described later, specific genes determine the synthesis of the V vs. C portions of a single Ig polypeptide chain. The basic form of the Ig molecule is a tetramer composed of two heavy and two light polypeptide chains connected by interchain disulfide bridges (Figure 22.3).

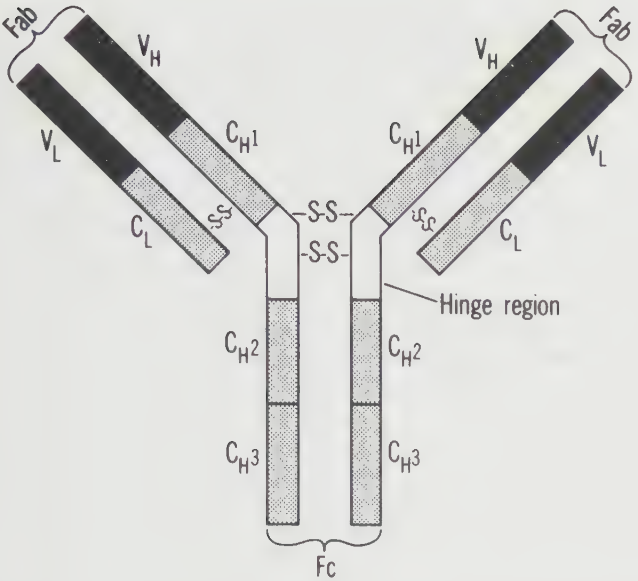

Figure 22.3. Schematic diagram of an antibody molecule. The location of the variable (V) and constant (C) domains of the light (L) and heavy (H) chains, the disulfide bonds (-S-S-), hinge region, antigen-binding fragment (Fab) and the crystallizable fragment (Fc) are shown.

Polymeric forms of the base structure of Ig molecules can occur depending upon the heavy chain contributions to the molecule. The heavy chain form is also the basis for defining Ig isotypes and to a large extent determines the biological function of the molecules. Ig molecules can exist in either a membrane-bound form or a secreted form. In mammals, the conversion from membrane-bound to secreted Ig is a result of differential processing of the heavy chain RNA transcript to the mature mRNA (Rogers et al., 1980).

Isotypes. The immunoglobulin (Ig) system of the chicken shares similarities with its mammalian counterparts, particularly with the existence of multiple isotypes. Chicken isotypes occur because of the existence of multiple heavy chain polypeptide chains that can contribute to Ig structure. Isotype heterogeneity is important since it is the carboxyl terminal portion of the Ig heavy chain that determines Ig effector functions. The opportunity to have the same antigen binding capacity distributed among several isotypes provides for a broader spectrum of different humoral immune responses, as well as effective regional immunity (e.g. mucosal surface protection, transfer of Ig to the embryo).

Three major chicken isotypes have received the most extensive research: IgM (17S sedimentation coefficient) is an effective crosslinking antibody appearing on the cell surface as 2(H+L) and in serum as a pentamer of the base structure (29S). The 7S Ig has been designated either as IgG or as IgY; this antibody is the predominant serum Ig and can also be transferred to the embryo. IgA is the predominant secreted Ig on mucosal surfaces and in bile. It can be associated with a secretory component (Watanabe and Kobayashi, 1974), and is particularly important in providing both G.I. tract immunity and protection of the eye from infections.

Two additional Ig isotypes have been investigated only recently. Some evidence has been obtained for the existence of chicken IgD, an Ig expressed early in B-lymphocyte differentiation (Chen et al., 1982). The possible existence of a chicken IgE immunoglobulin, an isotype significant in mammalian mast cell degranulation, is unclear at present. Isotype expression is developmentally regulated in the chicken with the sequence of heavy chain expression being μ, then γ. This developmental sequence corresponds to phylogenetic restrictions; some lower vertebrates possess only IgM with others having IgM and IgG but not IgA-like molecules (Litman, 1977). The chicken has predominantly one light chain type that is similar to the mammalian λ chain, based on partial sequence information. This λ chain relationship occurs despite the presence of an unblocked N terminal chicken light chain as in mammalian κ chains (Kubo et al., 1970). Ducks appear to have a major secretory Ig with a sedimetation coefficient of 5.7S compared with the 7S Igs of chickens and mammals (Häage and Ambrosius, 1984). The relationship of this duck Ig to other IgGs and IgAs has yet to be clearly established.

Allotypes. Isotypes of chicken Igs can show variation between individuals based on amino acid substitutions. Such chemical differences in polypeptide chains are termed allotypes and result from the inheritance of different alleles for gene(s) involving a particular isotype. While allotypes including those of poultry species have underlying chemical bases of definition, they are most frequently defined and monitored with the use of antisera prepared by allogeneic immunizations (Skalba, 1964). Allotypes have been identified both for chicken IgM (Pink, 1974; Foppoli and Benedict, 1977) and IgG (David, 1972; Wakeland and Benedict, 1975), and the genes encoding the IgM and IgG allotypes are known to be closely linked (Pink and Ivanyi, 1975). Most chicken allotypes have been identified through the use of genetic strains having combinations of antigenic determinants known as phenogroups. Unique serological specificities are used to distinguish between Ig allotypes (Ivanyi, 1975).

The ability to digest Ig molecules with proteolytic enzymes has provided one means of examining the site specificity of allotypic markers (Ch'ng and Benedict, 1981a). Papain digestion subdivides Ig molecules into the Fc (crystalizable fragment) portion and the Fab (antigen binding fragment) region of the molecule. In the case of IgG allotypes, some serological determinants are relatively resistant to papain digestion while others are destroyed by this process (Ch'ng and Benedict 1981a).

A survey of chicken IgM and IgG allotypes in pheasants, Japanese quail and turkeys revealed that only a limited number of chicken IgG allotypes were also present in these species. One question that remains is whether these avian species have evolved other IgG allotypic specificies that are absent from the chicken system.

In humans, one IgG1 allotype, nGm(1), occurs on some IgG1 molecules and on all IgG2 and IgG3 class molecules. The marker is an allotype for one class and an isotype for other Ig classes (Zaleski et al., 1983). This has led to the concept of isoallotypes where a serological specificity is part of a multiallele segregating system for one gene and is an invariant component among alleles for other genes. A related example may exist among avian species where the chicken G-1.7 serological specificity that is part of the chicken G-1 allotype system appears to exist in all pheasant and turkey sera (Ch'ng and Benedict, 1981b). If one considers these species as a group, G-1.7 is, in effect, a type of isoallotypic marker being an allotypic marker in the chicken and an isotypic marker in pheasants and turkeys.

Chicken IgG allotypes can be expressed at different concentrations. Predominant allotypes predicted by Mendelian inheritance are referred to as nominal allotypes. However, it is clear from the studies of Benedict and colleagues that chickens can express low levels of the 'incorrect' allotype on IgG molecules (Foppoli et al., 1978). These latent allotypes are not predicted by breeding and are at concentrations that apparently do not prohibit the preparation of alloantisera to nominal allotypic markers. While the regulation of nominal vs. latent allotypes is not understood in either mammals or avians, it is clear that chickens must possess the genetic information to express both nominal and latent allotypes and that inheritance involving some regulatory process determines whether a given allotype is expressed as a nominal or latent marker in a particular strain.

Allelic exclusion. In chickens, heterozygous for the IgM allotype (M1), individual B lymphocytes will normally express IgM molecules with only one of the two M1 specificities (Ivanyi and Hudson, 1979). The underlying basis of a similar phenomenon in mammals is the effective utilization of heavy chain genes from only one of the two homologous chromosomes in a given B-lymphocyte (Coleclough et al., 1981). This allelic exclusion of Ig expression occurs in chickens and leads to distinct populations of B-lymphocytes expressing one or the other M1 markers in M1 heterozygous birds (Ratcliffe and Ivanyi, 1979a).

Allelic exclusion is a crucial phenomenon to the integrity of the immune system. It ensures that a stimulated B-lymphocyte will secrete only one specificity of Ig, the one initially bound by antigen as the B-cell surface receptor. Otherwise, up to four different Igs could be synthesized by a single B-lymphocyte with possibly three of the antibody specificities being either extraneous or representing autoimmune specificities. At the very least, these antibodies would be unregulated in the B-cell receptor system.

Isotype and allotype suppression. Several studies have demonstrated that administration of anti-chicken IgM or anti-μ heavy chain to chick embryos prevented expression of not only IgM but also IgG synthesis (Kincade et al., 1970; Leslie and Martin, 1973). Since IgG expression results from a class switch from μ to γ during development and/or differentiation, suppression of μ production also prohibits γ heavy chain expression during subsequent differentiation.

Antiallotypic antibodies have also been used to suppress expression of one of two M1 allotypes in heterozygous chickens (Ratcliffe and Ivanyi, 1979b). Concomitant suppression of the G-1 allotype genetically linked to the suppressed IgM allotype was also observed. In effect, long-term suppression reflected the lack of Ig secretion by all B-lymphocytes that had committed to use a particular μ-γ containing parental chromosome.

Immunoglobulin genes and expression. Immunoglobulin genes including those in the chicken represent a structural type of gene family referred to as split-genes. With split-gene families, genetic elements or minigenes encoding discrete segments of a polypeptide chain are physically separated on a chromosome. Commitment to express the genes involves rearrangements that bring these minigenes into juxtaposition for transcription. The initial suggestion for multiple Ig genes encoding a single polypeptide chain came in the late 1960s (Dreyer et al., 1967) and the finding that the minigenes shifted chromosomal location upon B-lymphocyte differentiation was reported in 1976 (Tonegawa et al., 1976).

The chicken Ig gene family is arranged into two linkage groups, one containing the genetic elements encoding the heavy chain polypeptide, and a second linkage of light chain genes. Heavy chain genes are divided 5'-3' into $V_HD_HJ_H$ and C_H minigenes which correspond to DNA sequences encoding NH_2 to COOH amino acid positions of the polypeptide chain. Similarly, the light chain family contains 5'-3' V_L J_L and C_L genes also responsible for selected regions of the Ig light chain. As in mammals, the formation of complete Ig-encoding sequences at the DNA level occurs by the rearrangement of a remote variable region gene to a position immediately 5' to the composite D_H-J_H-C_μ for the heavy chain or J_L-C_L for the light chain. At the molecular level, allelic exclusion reflects the appropriate rearrangement of heavy chain and light chain genes on only one of the two available homologous chromosomes in a given B-lymphocyte. Ig gene rearrangement represents an important committed step in B-lymphocyte differentiation.

In mammals, Ig diversity is generated by heavy and light chain combinational associations, by use of multiple V_H D_H J_H and V_L J_L genes in factorial combinations to generate numerous composite IgH and IgL gene options, and by postrearrangement gene modification by somatic mutation. A surprising finding in the avian system is that chickens possess only one active V_L and J_L gene (Reynaud et al., 1985). Instead of using multiple V_L and J_L elements in various combinations, the chicken resolves the diversity challenge in a novel fashion. Diversity is generated in postrearranged sequences through a process of gene conversion involving the active V_L sequence and 25 pseudo V_L genes present in the chromosomal region (Reynaud et al., 1987a; Thompson and Neiman, 1987). The result is the generation of a large pool of unique light chain sequences all originating from a common precursor V_L-J_L-C_L gene. For the heavy chain, it also appears that a single subgroup of V_H genes encodes most chicken Ig heavy chains (Parvari et al., 1987). Since the Ig repertoire expansion process occurs in the bursa of Fabricius during ontogeny, it has been suggested that this organ may provide the necessary microenvironment for gene-conversion-dependent Ig diversity (Ratcliffe et al., 1987).

In mammals, heavy chain constant region genes are themselves subdivided into exons and introns (Sakano et al., 1979). The exons which correspond to polypeptide-encoding regions correspond approximately to individual Ig domains. Following Ig transcription, introns are removed by RNA splicing as are intervening regions between the rearranged minigenes. The resulting mRNA has contiguous $V_HD_HJ_HC_H$ or $V_LJ_LC_L$ corresponding sequences and a 3' end appropriate either for secretion of the subsequent Ig molecule or its placement in the membrane. Comparisons of the chicken heavy chain gene system and its transcripts with those of mammalians have been undertaken (Reynaud et al., 1987b).

Idiotypes. An idiotype represents a marker that identifies Ig molecules with specific antigen-binding regions. As such, idiotypes are distinct from either isotypes or allotypes. The same idiotype can be found on both IgM and IgG molecules. This represents the use of the same V_H sequences with both C_μ and C_γ in conjunction with the same light chain. Idiotypes are usually detected with anti-idiotypic antibodies. Idiotypic sites frequently correspond to one or two amino acid positions (Clevinger et al., 1980).

In the chicken, idiotypic markers have been detected on chicken antibodies against N-acetyl-glucosamine and p-amino-benzoic acid. The monoclonal antibodies used to detect these idiotypes also reacted with low percentages (< 1.0 percent) of circulating chicken lymphocytes using immunofluorescence (Chanh et al., 1982). With two other monoclonal antibodies produced against an avian B cell lymphoma, one designated cId-3 reacted with approximately one percent of B cells, while the second, CV_H-1 detected an Ig determinant on about one-third of these cells (Wright et al., 1987). These antibodies should prove useful in evaluating the generation of Ig diversity during B-cell differentiation.

Markers related to immunoglobulins. In the course of chicken immunoglobulin studies, investigators detected a normal serum protein of approximately 500,000 daltons molecular weight that was capable of precipitating antigen-antibody complexes. This protein has characteristics of a lipoprotein and migrates in the B-globulin region on gels. Euglobulin, as it has been designated, has recently been shown to be encoded by a polymorphic gene (*E-1*). Alloantisera produced using two inbred chicken lines have been used to identify two alleles of *E-1* ($E\text{-}1^a$ and $E\text{-}1^b$). These alleles segregate independently of Ig allotypes and MHC haplotypes (Ivanyi and Tempelis, 1980).

Hereditary agammaglobulinemia and autoimmunity. Strains of chickens exhibiting selective hypoagammaglobulinemia with dysgammaglobulinemia have been developed by two different research groups (Benedict et al., 1977; Brüggermann et al., 1967). These strains exhibit similar characteristics with deficiencies that resemble the human common variable immunodeficiency. Most birds possess elevated levels of serum IgM (dysgammaglobulinemia) with severely reduced levels of IgG. While the condition is clearly heritable, the exact mode of genetic control has yet to be established. Inheritance of this condition does not appear to be associated with either 7S Ig allotypes or B-haplotypes (Benedict et al., 1977; Fiedler et al., 1980). Associated with the selective immunodeficiency in the UCD line chickens are several autoimmune abnormalities (Montero et al., 1979). These include the production of hemolytic antibodies and antigammaglobulin activity. Such findings have led to the suggestion that abnormalities in regulatory leukocyte populations may lead to the resulting Ig abnormalities.

A second inherited condition leading to autoantibody production is hereditary spontaneous autoimmune thyroiditis (SAT) (Cole et al., 1968). SAT is characterized by thyroid infiltration by lymphocytes and the production of autoantibodies against thyroglobulin (Wick et al., 1982). The autoimmune obese strain, the MHC-matched control Special C strain, and the parent Cornell C strain population, have all been studied extensively by several research groups (DeCarvalho et al., 1981; Bacon et al., 1974; Wick, 1987; Gause et al., 1985). Three genetic components are thought to be involved in controlling the autoimmune phenotype: the MHC haplotype determining immune response properties, a non-MHC component influencing T-cell development and the production of regulatory T-cells, and genetic factor(s) influencing the thyroid as a target organ for autoimmune stimulation (Wick, 1987). Further discussion of SAT is included in Chapter 10.

One unifying feature of both genetic models described in this section is the prominent role of aberrant regulatory leukocytes leading to altered Ig properties. This suggests that genetic control of regulatory T-lymphocytes and macrophages may be an important consideration in genetic control of avian immune function.

THE T CELL RECEPTOR IN CHICKENS

One hallmark of the immune system is its ability to respond in a highly specific fashion to virtually any antigenic challenge. This specificity can be determined by either B lymphocytes or T lymphocytes. For B cells, the antigen-specific cell surface receptor is an immunoglobulin molecule. For T cells, the antigen-specific cell surface receptor, or T cell receptor (TCR), is major histocompatibility complex restricted (Davis and Bjorkman, 1988; Marrack and Kappler, 1987). The T cell receptors consist of two polypeptides joined into heterodimers, either α and ß or γ and δ, and a complex of nonpolymorphic proteins called

CD3. The α/ß heterodimer is present on most T cells, but the γ/δ heterodimer which appears earlier in ontogeny is present on only a small percentage of peripheral lymphocytes and the majority of skin dendritic T cells. In mammals, where the gene sequences are known, the T cell receptor primary sequence, gene organization, and mode of gene rearrangement have been found to be very similar to that of immunoglobulins. It is hypothesized that the T cell receptor interacts with an antigen-MHC protein complex, accounting for the T cell properties of antigen-specificity and MHC-restriction.

Most reports to date of chicken T cell receptors have come from the laboratory of Chen, Cooper, and coworkers (Chen et al., 1986; Chen et al., 1988; Bucy et al., 1988; Sowder et al., 1988). A monoclonal antibody, CT-3, reacted with three polypeptides (molecular weights 20,000, 19,000, and 17,000) on chicken T cells and was mitogenic for chicken T cells. It coprecipitated polypeptides of 49,000 and 38,000 molecular weight in T cell lysates. The antibody CT-3 therefore identifies the T3 complex in chickens. Studies of ontogeny showed that thymic cells acquired the T3/TCR complex after 5-6 days in the thymus, and migrated peripherally to the spleen one week later (Chen et al., 1986).

A monoclonal antibody, TCR1, was produced that identified a heterodimer resembling the mammalian γ/δ T cell receptor (Sowder et al., 1988). The chicken γ/δ T cell receptor appeared on the first wave of embryonic cells populating the thymus, and was present on a subpopulation of circulating T cells lacking CD4 (helper phenotype) and CD8 (cytotoxic phenotype) molecules. A much higher percentage (20 percent of circulating T cells and 30 percent of splenic T cells) of TCR1 positive cells was seen in chickens than in humans.

Use of monoclonal antibodies which identified the α/ß TCR (TCR2) or γ/δ TCR (TCR1) showed that these receptors are expressed on separate populations of T cells (Chen et al., 1988; Cihak et al., 1988). Most TCR2-positive cells in the thymus were positive for both CT4 or CT8, but in the blood or spleen TCR2-positive cells were positive for either CT4 or CT8, but not both. The TCR1-positive cells in the blood and thymus were negative for CT4 and CT8, but in the spleen were positive for CT8. The TCR1-positive and TCR2-positive cells also had distinctly different tissue homing patterns (Bucy et al., 1988). The TCR1-positive cells were located in the splenic sinusoids and intestinal epithelium, and expressed the CD8 homolog. The TCR2-positive cells were located in the splenic periarteriolar sheath and intestinal lamina propria, and expressed the CD4 homolog. The data support the concept that in chickens the TCR1- and TCR2-positive cells are separate subpopulations with distinct functions.

MAJOR HISTOCOMPATIBILITY COMPLEX GENES AND ANTIGENS

The major histocompatibility complex (MHC) of the chicken was initially identified by Briles et al. (1950) as an erythrocyte antigen or blood group system of extensive polymorphism. The gene locus symbol was changed, from the originally assigned *D*, to *B* to indicate that it was the second chicken blood group system discovered. A blood group locus discovered independently by Gilmour (1959) was later demonstrated to be identical to the *B* system of Briles. Early investigations which showed extensive multiple alleles of the *B* system, even in closed and inbred flocks, suggested a selective advantage of *B* heterozygosity for fitness and survival (Briles and McGibbon, 1948; Gilmour, 1959).

The mechanism by which the *B* system might influence fitness remained unknown until the discovery by Schierman and Nordskog (1961) that the *B* blood group system was linked to the major histocompatibility complex. In exchanges of skin grafts between birds of a relatively inbred strain having like and unlike blood types, grafts between birds of the same *B* type were consistently accepted, but those of different *B* blood types were rejected. This identified the linkage of *B* with the major skin graft locus. Additional evidence that *B* represented the chicken MHC was given by the identification of *B* as the graft-versus-host (GVH) splenomegaly locus (Jaffe and McDermid, 1962), the major GVH-chorioallantoic membrane (CAM) pock locus (Schierman and Nordskog, 1963), and the mixed lymphocyte reaction (MLR) locus (Miggiano et al., 1974). These genes affect the immune response, histocompatibility reactions, and response to disease.

Subregions of the chicken MHC. The complex nature of the chicken *B* system was not fully appreciated until the discovery of chromosomal recombinants in the chicken MHC. The first clearly defined recombinant was described by Hála et al. (1976) as a crossover between the MHCs of inbred lines CC and CB. This demonstrated, via genetic and serological analyses, that the chicken *B* system had a minimum of two loci, for which the designations *B-F* and *B-G* were proposed. The *B-F* locus was shown to determine histocompatibility and serologically determined antigens as well as controlling the GVH and MLR responses, and *B-G* was shown to determine only serological antigens (Vilhelmová et al., 1977). Ziegler and Pink (1975, 1976, 1978) combined biochemical characterization of MHC antigens with genetic analysis of *B* recombinants to develop a three locus model of the chicken MHC (Pink et al., 1977). This model of a chicken MHC composed of three subregions is still generally accepted today. The B-G antigen is located primarily on erythrocytes, the B-F antigen is on virtually all nucleated cells, and the B-L antigen is on monocytes/macrophages, B cells and activated T cells. The term haplotype is used to refer to the specific combination of alleles that an individual possesses for all the loci of the MHC.

To date, all recombinants of the chicken MHC have occurred between the *B-G* and the *B-F*/*B-L* subregions (Briles and Briles, 1977; Koch et al., 1983; Crone and Simonsen, 1987). Pevzner et al. (1978) reported on a recombination between genes encoding a humoral immune response and genes encoding the serologically determined antigens in the *B* complex. Although some have interpreted these data to represent a recombination between *B-G*/*B-F* and *B-L*, biochemical confirmation of this hypothesis has not been forthcoming. Additionally, restriction fragment length polymorphism analysis of these populations with a *B-Lß* probe does not demonstrate differences in MHC class II *B-Lß* genes associated with immune response genes as defined in Pevzner's study (Pitcovski et al., 1988).

The fortuitous location of genes encoding the erythrocyte antigen B (Ea-B) within the chicken MHC has allowed the use of blood typing with serological reagents as a method for identifying the chicken MHC by hemagglutination. Three different kinds of typing reagents can be used (Briles et al., 1982). Alloantisera which potentially detect both B-G and B-F are prepared by immunizations with whole blood between pedigreed birds compatible at blood group systems other than *B* (Briles and Briles, 1982). These sera are characterized by their reaction patterns with a panel of cells of known *B* type. Alloantisera specific for B-G or B-F antigens can be prepared by immunizations between birds of appropriate MHC-recombinant haplotypes that share a common genetic background or by immunizations with purified leukocytes or red blood cells and subsequent adsorptions with the appropriate cell type. A workshop held in 1981 in Innsbruck recommended a uniform nomenclature for standard and recombinant haplotypes and also designated a standard chicken strain to be used as the reference source for each of 27 known *B* haplotypes (Briles et al., 1982). Monoclonal antibodies to B-G antigens can also be used as typing reagents. Nomenclature for chicken MHC (B) antigens defined by monoclonal antibodies has been proposed by Longenecker and Mosmann (1981).

Chicken MHC antigens: B-G. The biochemical structure of the chicken MHC antigens has been studied by using polyclonal and monoclonal antibodies to visualize the antigens in complex mixtures of proteins and to isolate the antigens from cellular components for further analysis. The B-G (or chicken MHC class IV) antigens of the chicken are apparently highly immunogenic in the mouse, and this has allowed the production of several monoclonal antibodies specific for one, some, or all B-G alleles (Longenecker et al., 1979; Longenecker, 1982; Miller et al., 1982). Initially, it was hoped that a comprehensive panel of monoclonal antibodies could be developed for use as highly specific blood typing reagents. This has not been realized, except for a few haplotypes, but the anti-B-G monoclonal antibodies have been extremely valuable in studies of B-G expression and structure. Miller et al. (1984) used monoclonal antibodies directed against a widely shared B-G determinant to immunoprecipitate B-G antigens from erythrocyte membranes for analysis by using two-dimensional (2-D) gel electrophoresis. The patterns generated were varied among haplotypes from slight microheterogeneity to very complex patterns. This demonstrated a greater level of complexity for the *B-G* locus than had been previously recognized. The high resolution 2-D gel electrophoresis technique was also used to provide biochemical confirmation of recombination in the *B-G* subregion that had been originally identified by serological typing (Miller et al., 1988b).

Early reports on the number of chains and molecular mass of B-G antigens showed discrepancies which may be because of susceptibility of B-G antigens to proteolytic enzymes (Pink et al., 1977; Wolf et al., 1984; Miller et al., 1984). Salomonson et al. (1987) used enzyme inhibitors throughout their B-G isolation procedure, and found that reduced B-G consisted of 46-48 kd monomers, depending upon the haplotypes. Unreduced B-G existed as dimers of 85 kd or trimers of 130 kd. There was no evidence for glycosylation of the B-G antigen or changes in mass due to post-translational modification. Dimerization of B-G was shown to take place 20-30 min after synthesis of the monomers. Kline et al. (1988b) used two alloantisera to characterize the B-G antigens of the B^2 haplotype. One antiserum recognized a nonreduced 98 kd molecule which yielded, under reducing conditions, 40-kd and 44-kd molecules. Another antiserum recognized only 48-kd reduced molecules hypothesized to form a 90-kd homodimer under nonreducing conditions. The B-G antigens have no known immunological function, although they are highly immunogenic and appear to be necessary as an adjuvant for an efficient anti-B-F response (Hála et al., 1981).

B-F. The B-F or chicken MHC class I antigen is expressed on the cell membranes of almost all chicken cells including nucleated erythrocytes. Ziegler and Pink (1975, 1976, 1978) described an MHC antigen that was located on both peripheral blood leukocytes and erythrocytes and consisted of a heavy chain of 40-45 kd associated with a smaller polypeptide of about 12 kd (Figure 22.4). The smaller chain was not polymorphic, but the larger chain varied in weight in different haplotypes. Homology of chicken class I (B-F) antigen with that of mouse and human was shown by using partial amino acid sequences (Vitetta et al., 1977). Monoclonal antibodies have been produced to the chicken B-F antigen (Crone et al., 1985; Pink et al., 1985; Sgonc et al., 1987). These monoclonal antibodies were used to demonstrate that the expression of B-F antigen on chicken cells increases rapidly from no positive cells at hatch to adult levels (100 percent of bursal cells, red blood cells, and peripheral blood lymphocytes; 20 percent of thymus cells) at nine days

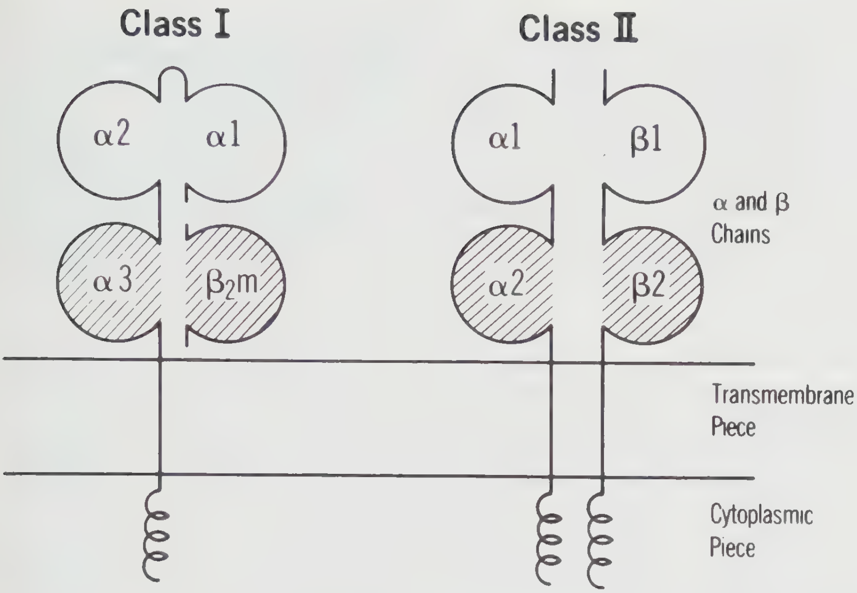

Figure 22.4. Schematic diagram of major histocompatibility complex class I and class II molecules. The α and ß chains, transmembrane and cytoplasmic pieces, and ß2-microglobulin (ß2-m) are shown.

post-hatch (Sgonc et al., 1987). Sequential immunoprecipitations of B-F antigens by monoclonal antibodies of different specificities indicated the existence of more than one *B-F* gene product (Crone et al., 1985). Kline et al. (1988a) used alloantisera against B-F antigens to identify 45 kd molecules on erythrocytes of embryonic chicks and two types of molecules on peripheral erythrocytes of adult chickens. The 'Type 2' molecules had multiple isomorphic variants and were associated with a 11-12 kd polypeptide. The 'Type 1' molecules had limited heterogeneity and were not associated with a smaller polypeptide.

The smaller peptide with which the B-F molecule is associated is assumed to be the avian equivalent of ß-2 microglobulin. A recent study (Skjoedt et al., 1986) characterizing the ß-2 microglobulin molecule of chickens and turkeys contradicted previous published reports (Winkler and Sanders, 1977; Lillehoj et al., 1982; Lillehoj and Poulik, 1982) but presented convincing evidence of its accuracy, based on binding to a ß2-microglobulin monoclonal antibody and recombining with the avian class I (B-F) heavy chain. The apparent molecular weight of this molecule, estimated from polyacrylamide gels, is approximately 14 kd. The 23 amino acid N-terminal residues were identical in chickens and turkeys, and bore a strong homology to mammalian ß2-microglobulin sequences.

B-L. The B-L or chicken class II antigen was identified by Ziegler and Pink (1976) as an antigen that was precipitated by an anti-B antiserum, was 30 kd, was not expressed on erythrocytes, and was not associated with ß2-microglobulin. Genetic and serological evidence indicated, respectively, that the B-L (or Ia-like) antigens were on the same chromosome as Ea-B and that they were analogous to MHC class II Ia antigens of mice (Ewert and Cooper, 1978; Ewert et al., 1980). Crone et al. (1981) demonstrated that the chicken B-L antigen has two separate species of molecules, now designated the α and ß chains. The α chain is 30-34 kd and the ß chain is 27-29 kd (Figure 22.4). These chains are noncovalently bound on the cell surface. The α-ß heterodimer matures in the cytoplasm in association with several basic invariant molecules (30-42 kd). The α and ß chains undergo complex N-glycosylation in the maturation process (Guillemot et al., 1986). The B-L antigens are primarily expressed on cells of the immune system: B cells, macrophages/monocytes, and activated T cells (Ewert et al., 1984; Hála et al., 1984; Guillemot et al., 1984; Peck et al., 1982). As in characterization of B-G and B-F antigens, monoclonal antibodies again have been valuable in isolating B-L antigens for detailed analysis. A non-MHC B lymphocyte alloantigen (Bu-1^b) has been shown to have a dominant effect in increasing the proportion of B-L positive cells in the bursa (Fredericksen and Gilmour, 1985).

MHC aneuploidy. Chickens that are aneuploid (trisomic or tetrasomic) for the MHC-bearing chromosome have been used to map the MHC, determine linkages with the MHC, and examine effects of MHC gene dosage on several biological systems (Bloom et al., 1987, 1988). A single hen having three *B* haplotypes was detected by routine blood typing. Trisomic lines, either with multiple segregating *B* haplotypes or with a homozygous *B* haplotype, were developed. Testcrosses were made and birds were blood-typed for the *B* haplotype and nucleoli-typed for the number of nucleoli by using acridine orange cytochemistry. Linkage studies demonstrated that the chicken MHC (*B* complex) and the ribosomal ribonucleic acid (rDNA) genes (nucleolar organizer region, NOR) are linked on a microchromosome of approximately size 16 (Bloom and Bacon, 1985). The excess rDNA genes appear to be inactivated in aneuploid birds, but the MHC genes on the same chromosome appear to be expressed in a dose-dependent fashion. Based upon both the final mean hemagglutination titres of disomic, trisomic, and tetrasomic cells with polyclonal antiserum, and on the number of each cell type needed to reduce by 50 percent the ability of the antiserum to hemagglutinate disomic erthrocytes, the aneuploids have a greater concentration of MHC glycoproteins on their erythrocyte surfaces than do disomic chickens (Delaney et al., 1987). Analysis by using flow cytometry of bursal lymphocytes to determine the concentration of B-L antigen on their surface demonstrated a stepwise increase of B-L antigen on trisomic and tetrasomic cells (Delaney et al., 1988). The finding that extra MHC copies are not inactivated has important implications for the feasibility of producing (by utilizing natural variation or genetic engineering techniques) chickens with increased copy numbers of the MHC genes to enhance health or production characteristics (Bloom et al., 1988; Lamont, 1989).

MHC gene isolation and cloning. The application of molecular genetic approaches to analysis of the genes of the chicken MHC has led to recent progress in understanding the unique genomic organization of this complex. Molecular genetic techniques to date have been used primarily in two areas of avian MHC research: isolating, cloning, and sequencing genes of the MHC, and molecular genotyping of MHC haplotypes by restriction fragment length polymorphism analysis.

Genes of all three subregions of the chicken MHC have been cloned. A *B-G* clone was selected from an erythroid cell λgt11 expression library by using antiserum against purified B-G antigen (Goto et al., 1988). This cDNA clone, designated bg28, was confirmed as a *B-G* subregion clone by using it as a hybridization probe. In Northern hybridizations, it bound specifically with erythrocyte mRNA. In Southern hybridizations, it mapped to the MHC-bearing chromosome based on its binding intensity to DNA from birds disomic or aneuploid for the MHC-bearing chromosome. It was further mapped to the *B-G* subregion based on its binding pattern to DNA of MHC recombinant birds. The hybridization patterns also suggested that multiple loci exist within the *B-G* subregion.

Six *B-F* genes have been isolated from a cosmid library derived from a CB line chicken of the B^{12} haplotype (Guillemot et al., 1988). The longest *B-F* cDNA clone, designated F10, was sequenced. The F10 clone was shown to be more divergent from mammalian class I molecules (equivalent avian and mammalian domains range from 24-52 percent amino acid sequence homology) than mammalian class I MHC molecules are from each other. Most residues involved in structural features of the antigen, however, were conserved.

The cDNA coding for chicken β_2M was isolated from a λgtll cDNA library from spleen and bone marrow cells, by screening with degenerate oligonucleotide probes (Skjoedt et al., 1988). This cDNA is not linked with the MHC, but β_2M and class I MHC molecules associate at the cell surface.

Several clones containing chicken *B-L* genes have been isolated (Bourlet et al., 1988; Guillemot et al., 1988; Béhar et al., 1988; Xu et al., 1989). Initial isolation of chicken class IIß genomic clones was carried out by hybridizing with a human DQß probe under low stringency conditions. The chicken class IIß genes showed 62-66 percent nucleotide homologies of genes encoding specific domains with the genes of corresponding domains from humans. The domains are the expected size (92-93 amino acids) and have the intrachain disulfide loop that is typical of the immunoglobulin supergene family (Bourlet et al., 1988). Subsequent isolations from libraries of other haplotypes (B^{12} and B^{6}) were done by screening with chicken class II (*B-L*) ß exons (Guillemot et al., 1988; Xu et al., 1989). Sequence homologies among MHC class II domains of chickens from different haplotypes range from 90-99 percent (Xu et al., 1989).

Molecular mapping of a large segment of the chicken MHC has revealed interesting details regarding the genomic organization of the *B* complex (Guillemot et al., 1988; Béhar et al., 1988). In contrast to currently sequenced mammalian MHCs, distances between genes of the chicken MHC are much smaller, with introns of about 100 bp. The close proximity of the *B-F* and *B-L* genes may account for the lack of recombinants identified between these two MHC subregions (Hála et al., 1988). There may even be overlapping of class I and class II regions. The *B* complex is tightly linked at its telomeric end to the nucleolar organizer. Several genes, whose function is currently unknown, are also located within the *B* complex. The general structure of MHC genes and antigens, however, is typical of the members of the immunoglobulin superfamily of genes and molecules. This suggests that the chicken MHC has also arisen by duplication and diversification of a primordial gene which was the progenitor of all immune-function related genes, such as those encoding immunoglobulins, T cell receptors, and MHC class I and II antigens.

Restriction fragment length polymorphism analysis of the MHC. The technique of restriction fragment length polymorphism (RFLP) analysis (see Chapter 24) provides a molecular genotyping approach which can be used to characterize differences in MHC genes among individuals (Lamont et al., 1987). Because the MHC has a well-established role in disease resistance, it should show RFLPs with direct effects on economic values and therefore be of value in a breeding program (Warner, 1986).

Chicken RFLPs have been generated with probes from the human MHC (Andersson et al., 1987) and from the chicken *B-L*, *B-F*, and *B-G* subregions (Chaussé et al., 1989; Lamont et al., 1987b, 1988; Gerndt et al., 1988; Hála et al., 1988; Miller et al., 1988a; Pitcovski et al., 1989). The use of RFLP analysis, as a biochemical characterization of the

MHC, is a valuable addition to serological analysis of the MHC. Caution must be exercised in interpreting restriction patterns, however, because fragment lengths only indicate the distance between two restriction sites, and do not give any information about the intervening sequences. Also, a single base change which may alter a restriction site may not encode a different protein product and conversely, some base changes which may significantly alter MHC antigens may not be located in common restriction sites and therefore not be identified by RFLP analysis. Thus, although different RFLP patterns indicate different DNA sequences, identical RFLP patterns do not ensure identical DNA sequences. The use of RFLP analysis is nonetheless valuable to examine subregions and genotypes for which producing serological reagents has been difficult, to analyze MHC chromosomal recombinants, and to categorize newly identified genotypes. In the future, standard nomenclature should be defined for RFLP analysis of MHC haplotypes, as was previously done for serological analysis (Briles et al., 1982).

MHC in other avian species. There has been relatively little study of the MHC in avian species other than the chicken. Graft-versus-host (GVH) reactivity, measured by the chorioallantoic membrane test, segregates in turkeys (Shoffner, 1964). Two independently segregating loci control the turkey (GVH) splenomegaly response (Cherms, 1977). An early study was unable to demonstrate that turkey erythrocytes have blood group markers of the MHC (Palmer and Nordskog, 1980). A recent report, however, indicates that several chicken alloantisera for MHC antigens differentiated histocompatibility types in the turkey (Ellis et al., 1988). Recently, a blood group in ring-necked pheasants has been identified which may represent the major histocompatibility complex of that species (Jarvi and Briles, 1988).

MINOR HISTOCOMPATIBILITY LOCI

In addition to the major histocompatibility complex, there are several minor histocompatibility loci (summarized in Pazderka et al., 1975). Minor histocompatibilities are associated with the *C* blood group locus and with the W and Z sex chromosomes. Based upon graft survival data, up to 12 minor histocompatibility loci may exist. The effect of these loci on compatibility is much less marked than that of the MHC.

GENETIC CONTROL OF IMMUNE RESPONSE: MHC CONTROL

One of the very important functions of the chicken major histocompatibility complex (MHC) is the regulation of cellular communication in the immune response. The cell surface protein products of the MHC, which are unique to each genetically unique individual, serve to distinguish 'self' from 'nonself'. This allows immune reaction to occur against foreign antigens of an almost infinite spectrum, to protect each organism's identity while simultaneously preserving the organism from self-destruction. The control of the MHC over communication events can occur at a minimum of two levels: molecular interactions at the cell surface, and cellular cooperation in an immune response (Dietert, 1987b). The MHC cell surface glycoproteins interact with both the antigen and with complementary structures of other immune cells. This communication generates an immune response which is specific for the inducing antigen.

MHC restriction of cellular interaction. In order for some of the different cell types of the immune system to communicate effectively, they must share at least one MHC haplotype. This phenomenon is known as MHC restriction. The MHC restriction may function at the level of interactions between mature cells in the immune response or at the level of immune cell differentiation (Vainio and Toivanen, 1987; Vainio et al., 1987).

The cyclophosphamide (CP) model was used to determine the genetic requirements for T-B cell interaction in the chicken. Administration of CP in the neonatal period causes a permanent depletion of the B-cell compartment, but T cells are only moderately affected and T cell function is normal 3-4 weeks after CP treatment. Transferred allogeneic bursal cells can repopulate the CP-treated host, but express only MHC products of the donor genotype, not that of the recipient. Therefore, antigen-presenting cells and T cells share MHC antigens with each other, but not with the grafted allogeneic bursal-derived cells. In this model, antibody production to a T-cell-independent antigen (*Brucella abortus*) could be restored by allogeneic or syngeneic bursal cells, but only syngeneic cells could restore antibody response to a thymus-dependent antigen (sheep red blood cells) (Vainio et al., 1983; Toivanen et al., 1974). The use of MHC-recombinant chicken lines revealed that the class II (B-L) antigens alone were the necessary restriction elements in the T-B cell cooperation for antibody production (Vainio et al., 1984).

Identity at the MHC has been found necessary for the T-B cell cooperation needed for generation of germinal centres in the spleen (Toivanen and Toivanen, 1977). Again, the use of MHC-recombinant chicks has pinpointed the restriction element to the class II region of the MHC (Vainio et al., 1984).

The interaction of antigen-presenting cells and T cells can be evaluated by the *in vitro* proliferation of T cells in response to specific antigen in the presence of antigen-presenting cells. By using MHC-recombinant chicken lines, it was demonstrated that the B-L (class II) gene products are the restriction elements for antigen-presenting-cell-T cell interaction (Vainio et al., 1988).

In addition to restricting the interaction of different types of immune cells with each other, the MHC also restricts T cell cytotoxic reactions with some virus-infected and/or transformed cells. When cytoxic T cells were combined with reticuloendotheliosis virus (REV) infected cells, significant cytotoxicity occurred only with syngeneic effector and target cell combinations (MacCubbin and Schierman, 1986). The effector cell responsible for the MHC-restricted cell-mediated cytotoxicity was characterized as an Ia-positive T cell with antigen-restricted cytotoxic properties (Weinstock and Schat, 1987). The MHC-restricted and virus-restricted cytotoxic cells may represent an important immune surveillance mechanism to limit proliferation of virally induced tumors in chickens (Schierman and Collins, 1987).

MHC and immune response: alloreactivity. Genetic control of immune response has been associated with the MHC in chickens by studying differential responses of birds possessing different MHC haplotypes and by using a variety of assays of immune responsiveness. The populations used have consisted of several types of genetic structures, including MHC-recombinant haplotypes, *B*-congenic lines, sublines differing at the MHC, and lines in which MHC haplotypes are segregating. Selected examples of MHC genetic control of alloreactivity and physiological immune responses will be given.

The earliest studies of the chicken MHC, which confirmed its linkage with the *B* blood group locus, were studies of alloreactions. The *B* complex was shown to be linked to genes controlling skin graft rejection (Schierman and Nordskog, 1961), graft versus host (GVH) splenomegaly (Jaffe and McDermid, 1962; Simonsen et al., 1977) and mixed lymphocyte reactions (Miggiano et al., 1974). These studies confirmed that the chicken MHC controlled T cell activation by allogeneic cells in a manner similar to that known for the mouse and human.

MHC and immune response: physiological role. Alloreactivity, however, is an artificial albeit useful assay system. A bird's immune system is unlikely to encounter cells from another bird, except perhaps in the case of accidental injury during artificial insemination. The actual important physiological role that the MHC plays is in serving as a restriction element for T cell recognition of antigen (Crone and Simonsen, 1987). The T cell recognizes an antigen in the context of its own MHC. The only case in which a T cell recognizes antigen alone is probably when the antigen is an MHC antigen. As described previously in this section, the class II MHC molecules are restriction elements for T cell-B cell and T cell-antigen presenting cell cooperation. The MHC also restricts T cell cytotoxicity of virus-infected cells.

Via its role as a restriction element or via specific immune response genes, the MHC has profound effects on genetic control of immunoresponsiveness. The humoral immune response (antibody production) against a variety of antigens has been shown to be linked to the chicken MHC. Immune response to synthetic polypeptides such as (T, G)-A--L (Günther et al., 1974), GAT (Benedict et al., 1975), and GT (Pevzner et al., 1979) have been demonstrated to be under MHC control in the chicken. Antibody titres to other soluble antigens (bovine serum albumin, BSA) and to cellular antigens (*Salmonella pullorum* bacterin) are also associated with the chicken MHC (Pevzner et al., 1975). Additionally, total serum IgG levels were shown to be under genetic control, partially associated with the *B* complex, but more significantly associated with the *Ir-GAT* locus (Rees and Nordskog, 1981).

Assays of cell-mediated immunity can also be used to evaluate genetic control of immunoresponsiveness. The delayed wattle reaction (DWR) is one such measure of cell-mediated immunity in the chicken (Klesius et al., 1977). The antigen used can either be a polyclonal activator of T cells, such as phytohemagglutinin (Taylor et al., 1987), or it may be a specific antigen, such as *Staphylococcus aureus* antigen (Cotter et al., 1987) which is an important pathogen of poultry. The DWR to *S. aureus* antigen showed an interaction between *B* complex and sex. In males, the *B* genotype significantly influenced the maximum response level of the DWR; however, in females, no *B* genotype effect was detected. The sex-influenced heterosis is hypothesized to be due to hormonal differences.

Although the complement system does not exhibit the memory and specificity of the humoral and cell-mediated immune system, complement proteins are extremely important in the immune reaction as a means to destroy invading pathogens. Complement components which are activated by antibodies or bacteria can lyse bacteria by destroying their cell walls. The levels of total serum hemolytic complement have been associated with the MHC in chickens (Chanh et al., 1976).

One very powerful tool in the genetic analysis of MHC control of immune response is the use of *B*-congenic lines of birds. These lines differ from each other only in the microchromosome, or a portion of it, which bears the MHC. Because the genetic background of these lines is uniform, any differences detected in immune response are attributable to the MHC-bearing chromosome. Congenic lines have been developed by several laboratories including the Regional Poultry Research Laboratory at East Lansing, Michigan. Analysis of partially developed (2-5 backcross generations) $15I_5$-*B* congenic lines showed no significant difference in immunoglobulin M or G levels or differential blood cell counts, but did show significantly different hematocrits and hemolytic complement levels (Shen et al., 1984). There were also *B*-genotype associated differences in antibody responses to sheep red blood cells, *Brucella abortus* antigen, and infectious bursal disease virus vaccine (Bacon et al., 1987). Chemotactic activity of chicken blood mononuclear leukocytes, and activity and recruitment to the peritoneal cavity of macrophages were also shown to differ among the *B*-congenic lines (Qureshi et al., 1986, 1988). All these examples illustrate the diverse facets of the immune response over which the MHC exerts genetic control.

The MHC has also been associated with various production traits such as mortality (Briles and McGibbon, 1948; Gilmour, 1959), and MHC allelic frequencies have been shown to be altered significantly by selection for production traits (Gavora et al., 1986; Simonsen et al., 1982; Lamont et al., 1987a). The mechanism by which the MHC may influence these traits is not known. The effects may be indirect, via influence on immunocompetency and disease resistance (see Chapter 33), or the effects may be more direct and based upon the role of the MHC in mediating cellular communication.

GENETIC CONTROL OF IMMUNE RESPONSE: NON-MHC CONTROL

Immune response differences in MHC-identical birds. The MHC has been the primary set of genes identified with major genetic control of immune response and disease resistance. There are, however, many genes located outside of the MHC that also regulate immune response. The most obvious examples of non-MHC gene control of immunoresponsiveness are found in those cases where birds possess identical MHC genes, but differ significantly in some aspect of immune function. Palladino et al. (1977) compared the response of two inbred lines, both homozygous for B^2, to four antigens and a contact sensitizer, oxazalone. One line was superior in delayed type hypersensitivity and in antibody responses to three of the antigens plus oxazalone. The other line gave higher responses to one of the four antigens, implying the existence of at least two non-MHC gene loci controlling immune response.

Comparisons were made of graft vs. host response in three lines that were all homozygous for B^{15} but in a background containing either an autosomal dwarf gene, a sex-linked dwarf gene, or no dwarf genes (Erf et al., 1987). There were significant differences between the strains in the GVH response, and interaction of genotype with age was observed. Strain differences in hormonal levels are hypothesized to influence the GVH response in this study.

A selection experiment was carried out within a homozygous B^1B^1 blood type for high and low antibody response to *Salmonella pullorum* (Pevzner et al., 1981). Maximal divergence in antibody titres was reached in the third generation of selection. The response to selection suggested a non-MHC polygenic control for *S. pullorum* antigen response.

Genetic selection for antibody response to sheep erythrocytes. There is a great deal of interest in identifying a genetic marker which can be used for selection for general immunoresponsiveness (Gavora and Spencer, 1983; Nordskog, 1983; van der Zijpp, 1983a). One system which has been examined in chickens is selection based upon antibody response to sheep red blood cells (SRBC), a complex nonpathogenic T-cell dependent antigen. A similar classic experiment in selection for SRBC antibody response in mice was conducted earlier by Biozzi et al. (1979). Two divergent selection experiments were conducted in chickens in which the birds were selected for either high or low antibody response five days postimmunization or for persistence or nonpersistence of antibodies between five and 21 days postimmunization (Siegel and Gross, 1980). There was evidence of additive and nonadditive genetic variation for 5-day titres and additive genetic variation for persistence of antibody titre. There was no response to selection for nonpersistence of SRBC antibodies. Other studies demonstrated that the details of the immunization procedure are important in experiments of this type. Different routes (intravenous, intramuscular, intraperitoneal) of immunization significantly affected the total SRBC antibody titres (van der Zijpp et al., 1986). Concentration of SRBC used for immunization can yield different types of results. High doses yield the highest titres, but low doses may be more effective in detecting genetic differences in immune response (Gross, 1979).

The SRBC antibody response has also been used as a model system for genetic analysis of humoral immune response in the chicken. Measurement of total, 2-mercapto ethanol (2-ME) sensitive, and 2-ME resistant titres at days 3, 7, 10, and 13 postinjection in White Leghorn chickens gave heritability estimates between 0 and 0.5 for all parameters (van der Zijpp and Leenstra, 1980). Additive genetic correlations between total antibody titres after primary and secondary immunizations were negative on days 5, 7, and 10 postsecondary injection, suggesting that improving overall antibody responsiveness via selection for either primary or secondary response will be difficult (van der Zijpp et al., 1983). No correlation was seen between the SRBC antibody response on days 3, 7, and 14 days postinjection and the phytohemagglutinin (PHA) wingweb response, a measure of T cell-mediated immunity (van der Zijpp, 1983b). This suggests that both antibody production and cell-mediated immunity measures need to be included in selection for general immunocompetence.

Genetic selection for early antibody production. Genetic control of immune response has also been investigated by selecting for early development of immunocompetence, as measured by antibody production. Early immunocompetency is especially important to control disease in the vulnerable neonatal period and during the short time span of commercial broiler production. After four selection cycles, the average titre index of broiler lines selected for high combined response to *Escherichia coli* and Newcastle disease virus vaccines was 68 percent higher than lines selected for low antibody response to the vaccines (Pitcovski et al., 1987a). Realized family heritabilities were 0.72 and 0.67 for two replicates. The line selected for high *E. coli* and NDV antibody response also had a higher antibody response to SRBC (but not *Brucella abortus*), higher levels of IgG (but not total Ig), greater bursa weight, greater numbers of spleen plaque-forming cells to *E. coli*, higher delayed type hypersensitivity response, higher T cell mitogenic response, and more rapid clearance of bacteria from the circulatory system (Pitcovski et al., 1987b). Thus, selection for early high antibody levels significantly altered several other immune system parameters. There was, however, no genetic correlation found in this study or another between the response to the two antigens (Pitcovski et al., 1987a; Soller et al., 1981).

Genetic control of mitogenic response. In response to stimulation with an antigen, a small antigen-specific set of immune cells is stimulated. In contrast, mitogens are substances that stimulate a large proportion of immune cells to undergo blast transformation and to proliferate. Mitogens are therefore used to measure the proliferative capacity of certain cell types (such as T lymphocytes) as an indicator of general cellular immunoresponsiveness. Phytohemagglutinin (PHA) and concanavalin A (Con A) are mitogens known to stimulate T cells in avian species (Greaves et al., 1968; Weber, 1975). Genetic control of PHA and Con A response has been demonstrated in chickens (Morrow and Abplanalp, 1981). Initially, a study of Con A response in two inbred lines led to the hypothesis that a single dominant gene (designated *Mr1* for mitogen response 1) controlled ability to respond (Miggiano et al. 1976). Investigation of additional genetic lines led to the proposal that at least two loci control Con A response in chickens, because genetic complementation between different low responder lines generated uniformly high responder F_1 birds (Pink and Miggiano, 1977). The low responder phenotype, regardless of locus, was due to an intrinsic inability of the T cells to respond to Con A by differentiating into growth factor-responsive blast cells (Pink and Vainio, 1983). Although the previous study did not show complementation in *in vitro* co-cultures of low responder cell types, another study did (Morrow and Abplanalp, 1984), implying that gene defects may occur in different aspects of the mitogenic mechanism and that more than two genes may influence Con A response in chickens. Separate genetic control of response to Con A and PHA has been demonstrated by the discovery of lines with comparable Con A response and variation in PHA response (Lassila et al., 1979). Control of the PHA response seemed to involve non-MHC genes; this was also confirmed by others (Lee and Bacon, 1983) who demonstrated PHA response differences in birds which were identical for the MHC. More recent genetic analysis with other inbred lines has confirmed the separate genetic control of PHA and Con A response in chickens, and indicates that one of the loci controlling Con A response is linked to the MHC (Knudtson et al., 1989).

Complement polymorphisms. Although total hemolytic complement activity in the chicken (Chanh et al., 1976) and genetic polymorphisms of several of the complement system components of mammals have been associated with the MHC (Porter, 1985), no complement component polymorphisms have yet been linked to the avian MHC. Multiple molecular forms of factor B have been identified in the chicken and their genetic control was not linked to the MHC (Koch, 1986).

Genetic control of lymphokines and cytokines. Lymphokines and cytokines are soluble factors produced by cells of the immune system that have biological effects on other cells of the same or a different type. These important molecules of the immune system are just beginning to be characterized in avian species. Interleukin 2 (IL-2) is a lymphokine produced by activated T cells which stimulates the proliferation of activated T lymphocytes. Lines of chickens have been shown to differ in IL-2 activity (Knudtson and Lamont, 1989; Knudtson et al., 1989; Schauenstein et al., 1982). The genetic control of IL-2 activity is distinct from that of mitogen response and is hypothesized to be under the control of at least two genes, one of which may be MHC-linked (Knudtson et al., 1989).

Biological mechanisms of genetic control of immune response. In most cases of non-MHC genetic control of the immune response, the exact biological mechanism involved is not known. Most immune phenomena are complex processes. As an example, although antibodies may be produced by B cells, the production and the amount, affinity, and kinetics of their production are subject to interaction with other cell types. Macrophages process the antigen and T cells may function as helper cells and produce necessary lymphokines. Signals between cells can be via direct physical interaction or via soluble factors. Signals must be transferred from the cell surface to appropriate subcellular organelles for response. Additionally, the cells must differentiate and interact in an appropriate microenvironment to support their response. Thus, genetic regulation of immune response can be expressed at many different biological levels.

IMMUNOGENETICS AND DISEASE RESISTANCE

Efficient functioning of the immune system is one important aspect in resistance to disease in poultry. Therefore, a complete understanding of the genetic aspects of immunity will be valuable to aid in selection for genetic resistance to disease (see Chapter 33). This understanding should include knowledge about genetic correlations between different facets of immune function (chemotaxis, phagocytosis, cytokine production, humoral immunity, cell-mediated immunity), heritabilities of each immune function, correlated responses of immune and production traits, genetic control of lymphokine production and markers delineating functionally distinct subpopulations of immune cells, and the detailed structure and function of the MHC.

Because of the multiplicity of pathogens to which a bird is exposed, selection for general disease resistance by indirect selection on immune response traits may be the best long-term strategy (Gavora and Spencer, 1983; Nordskog, 1983; van der Zijpp, 1983a). Some selection experiments based on immune response traits have already yielded information about correlated responses of immunogenetic selection on general disease resistance capabilities. Lines selected for high SRBC antibody production were more resistant to *Mycoplasma gallisepticum*, *Eimeria necatrix*, splenomeglia virus, and feather mites than the low antibody selected line; however the high line was more susceptible to *E. coli* and *S. aureus* infection (Gross et al., 1980). Selection for early high antibody response to *E. coli* and Newcastle disease virus vaccines produced a population more resistant to *E. coli* challenge than the low antibody selected line (Pitcovski et al., 1987a). Many examples exist of alteration of disease resistance in populations based upon MHC differences. Information is also now becoming available about genetic correlations between different facets of the immune response (Cheng and Lamont, 1988; van der Zijpp, 1983b) which will be useful in developing strategies for applying immunogenetics knowledge in a practical fashion to improve immune response and disease resistance in poultry populations.

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Chapter 23. MOLECULAR GENETICS

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Historically, chickens have played a more significant role in molecular genetics than in most areas of biology. The egg white protein genes were among the very first to be cloned using recombinant DNA techniques because of the availability of large amounts of biological material due to the genes' high levels of expression. Their analysis added important general concepts to the understanding of gene structure and function in higher organisms. They were also the first hormone-inducible genes to be cloned and this led to some of the first detailed analyses of regulatory sequences adjacent to structural genes, the nature of the hormone receptor proteins and their genes, and the analysis of interactions between the receptor complex and DNA. In these areas, work on chicken genes is still at the forefront of molecular genetic research in higher organisms. More recently analysis of hormone regulation has extended to egg yolk protein genes and other genes synthesized in the liver, adding further dimensions to analysis of the interaction between hormone inducibility and tissue specificity.

Similarly the skeletal muscle structural genes of the chicken were in many cases cloned before those of mammals, again mainly due to the availability of large amounts of the tissue expressing the genes. In other cases though, chicken genes have been cloned using mammalian homologues (or their sequences) to give information on evolutionary relationships.

More recently, with the possibility of producing genetically-manipulated transgenic chickens (see Chapter 24), it has become important to identify the genes controlling commercially important characteristics such as muscle growth, reproduction, and disease resistance (Bulfield, 1985). These complex characteristics are important topics for analysis in their own right, using recombinant DNA techniques and transgenic animals, as aspects of development as well as creating the possibility of genetical improvement by genetic manipulation techniques.

The problem in reviewing the current state of molecular genetics of any topic is that the field is moving so fast that a mere catalogue of data will undoubtedly be out of date before publication. In this chapter, the major groups of chicken genes that have been cloned are reviewed, pointing out their contribution to understanding of the molecular genetics of higher organisms in general and the directions they indicate for future research. An abbreviated list of cloned genes from the EMBL and GenBank lists (as of July, 1988) is shown in Table 23.1. This listing might encourage the reader to do his own search to bring it up to date. In this chapter, certain groups of cloned genes that are discussed in other chapters have been excluded; these include major histocompatibility complex genes, retroviral insertion and oncogenes, and certain other groups such as repetitive DNA sequences.

Study of the major groups of chicken genes has contributed greatly to understanding of the molecular genetics of higher organisms in general over the last ten years. It appears that there will in the future be a change of emphasis towards cloning and analysis of genes controlling major commercially important traits and their utilization in transgenic animals.

EGG WHITE AND EGG YOLK PROTEIN GENES

Gene Structure

In any discussion of cloned chicken genes, precedence must be given to the egg white protein genes (see review by Sippell, 1984) and especially to ovalbumin. The four major egg white gene products produced in the oviductal tubular gland cells (ovalbumin, conalbumin/ovotransferrin, ovomucoid and lysozyme) account for over 70 percent of the protein. About 70 percent of oviductal mRNA consists of about ten abundant RNA species; 85 percent of the other 12,000-15,000 diverse RNA species are common to both oviduct and liver (Axel et al., 1976).

Ovalbumin. Ovalbumin is the major protein of egg white, comprising about 60 percent of the total. The abundance of its mRNA in the oviduct of the laying chicken facilitated RNA purification by chromatography and cDNA synthesis (Woo et al., 1976, 1977) or by polysome immunoprecipitation with a specific anti-ovalbumin antiserum (Humphries et al., 1977), followed by cloning in a plasmid in *E. coli* (Humphries et al., 1977; McReynolds et al., 1977). These favorable circumstances placed ovalbumin among the first three eukaryote genes to be cloned (along with silk moth fibroin and rabbit ß-globin).

The use of this ovalbumin cDNA to isolate the chromosomal gene from the chicken genome immediately demonstrated that the coding sequences residing in a 1.87 kilobase (kb) mRNA were spread over 7.5kb of genomic DNA split into eight pieces (Breathnach et al., 1977; Dugaiczyk et al., 1978; Garapin et al., 1978a, 1978b; Mandel et al., 1978). Again ovalbumin was among the earliest genes for which these intervening genomic sequences (IVS; introns) had been found, a feature startling at the time but which is now known to be a characteristic of almost all eukaryotic genes. At that time ovalbumin was the gene with the largest number of introns that had been sequenced and this allowed comparison of sequences at exon-intron boundaries. It was discovered that at the 5' end of the introns was the G-T dinucleotide, and at the 3' end the A-G dinucleotide (Breathnach et al., 1978). This rule has been found throughout eukaryotic genes. It was soon found that the introns were transcribed into RNA in the nucleus but that the intronic (noncoding portions) were selectively spliced out and degraded (Roop et al., 1978).

Ovalbumin was also among the first genes for which polymorphism in restriction sites within introns was found for both an EcoRI site and Hae III site (Lai et al., 1979). This introduced a novel type of allelic variation within a locus which was 'silent', having no effect on the sequence of the protein. These variants were the earliest restriction fragment length polymorphisms (RFLPs) which are now so commonly reported in eukaryotic genomic DNA.

The 5' end of the chromosomal ovalbumin gene also received early attention with the expectation that it would contain regulatory sequences, especially those controlling hormone induction (see below). Once the 5' end of the gene had been sequenced, a sequence 5'-TATATAT-3' was discovered about 30 nucleotides before the 5' end of the mRNA, which strongly resembled both the Pribnow-box of prokaryotic promoters and the Hogness-box of histone genes (Gannon et al., 1979). When this 'TATA-box' was compared with that of the rabbit ß-globin, mouse ß-globin major and adenovirus-2 major late genes, it was found to be remarkably conserved both in sequence and position relative to the 5' end of the mRNA (Gannon et al., 1979). This TATA-box has now been generally found about 34 nucleotides prior to the 5' end of most eukaryotic genes.

A detailed comparison of the 5' end of the ovalbumin gene cloned from an expressing tissue, oviduct, and a nonexpressing tissue, erythrocytes, shows no alteration in sequence between the two tissues (Gannon et al., 1980). Therefore all the sequences necessary for expression or nonexpression of the gene are present in all tissues and tissue specific regulation must be controlled by *trans*-acting factors. The 5' end of the gene has been extensively analyzed from the point of view of hormonal induction of the gene and this is dealt with later.

The 3' end of the ovalbumin gene has also been extensively analyzed. A hexanucleotide sequence AAUAAA is located 11-30 nucleotides upstream from the poly(A) tail of eukaryotic mRNAs and functions as a signal for polyadenylation. Multiple polyadenylation signals have been found for the ovalbumin (Le Meur et al., 1984) and the X and Y genes (Le Meur et al., 1981) producing RNAs of different lengths. In all cases there is a major 'classical' polyadenylation signal which is the major termination signal and is responsible for the major class of RNA.

When analysis of the natural ovalbumin gene was extended beyond the 7.7kb transcriptional unit to a 46kb region, an unexpected finding was made. Two other genes, called X and Y, with sequence homologies to ovalbumin but of unknown function were found, orientated in the same direction as ovalbumin and also under hormonal control (Royal et al., 1979), although expressed at much lower levels than ovalbumin (X one percent, Y ten percent; Colbert et al., 1980). Sequence analysis of the X and Y genes showed conservation of numbers of introns, splice sites, and nucleotide protein coding sequences (76 percent X:ovalbumin, 73 percent Y:ovalbumin; Heilig et al., 1980, 1982) indicating that the three genes have evolved from a common ancestor by duplication but have remained very closely linked in the genome. The introns and the 3' untranslated regions have diverged much more, once again a finding now common in families of eukaryotic genes.

The DNA sequence of the ovalbumin, X, and Y genes allowed a predicted protein sequence to be determined and a search for relatives in the protein sequence databases. This revealed a surprising although fairly distant relationship between these three genes and two human plasma protease inhibitors, antithrombin-III and alpha$_1$-protease, forming a new protein superfamily (Hunt and Dayhoff, 1980). Once it was cloned and sequenced, α_1-antitrypsin was added to this superfamily, all the members sharing amino-acid sequence homologies of around 30 percent (Leicht et al., 1982: Long et al., 1984). The function of ovalbumin in egg white had previously been unknown. These sequence similarities to protease inhibitors point to a function compatible with the role of egg white in embryonic development and the known function of the other three major egg white proteins, ovomucoid (protease inhibitor), conalbumin (iron-binding bacteriostat), and lysozyme (bacteriocide).

The ovalbumin, X, and Y genes are not only coordinately expressed in the chicken oviduct; they are also in the same 100 kilobase chromosomal domain sensitive to DNase I (Lawson et al., 1980, 1982). At the 5' and 3' borders of this domain are found members of the CR1 family of repetitive sequences (Stumph et al., 1983). There was however no evidence of specific DNA rearrangements involved in the expression of the gene although the 3' end of the region is undermethylated in the oviduct compared with other tissues (Stumph et al., 1983). Analysis with micrococcal nuclease demonstrated that the coding region of ovalbumin in hormone-treated chickens was more sensitive to enzymic digestion than inactive flanking sequences or coding sequences from hormone-withdrawn chickens. There were some sites upstream from the gene that were sensitive to nuclease, irrespective of hormone status of the birds, although these sites were resistant to digestion in nonoviductal cells. Therefore sites upstream from the ovalbumin gene are available for digestion prior to hormone treatment whereas the coding region is only available after hormone treatment (Anderson et al., 1983).

In the terminal 3' 2kb of the ovalbumin pre-RNA coding sequences and 14kb immediately downstream, there are four major and two minor DNase I hypersensitive sites in oviduct chromatin (Bellard et al., 1986). One of these major sites is hormone-dependent and encompasses both the major and minor polyadenylation sites and signals (Bellard et al., 1986).

The position of the ovalbumin gene in the nucleus differs between expressing oviductal cells and nonexpressing erythrocyte cells. The transcriptionally active ovalbumin gene is preferentially associated with the nuclear matrix whereas the inactive gene is not, and this must have significant implication in control of gene expression (Robinson et al., 1982).

Although the active phase of research in the structure and expression of the ovalbumin gene has now moved on to the detailed analysis of hormone regulation, several important concepts of eukaryotic gene structure and function were introduced from the analyses reviewed above; these include the existence of introns, the GT-AG rule for their splicing, restriction fragment length polymorphisms in introns, the TATA-box, the role of *trans*-acting factors in tissue-specific expression, the existence of tandem duplicated sibling genes, the organization of chromosomes in gene expression, and finally, a physiological role for ovalbumin, the first success for reverse genetics in solving a biological riddle.

Conalbumin. Conalbumin is the major iron-binding protein of egg white constituting about ten percent of the protein. It is presumed to be a nutritional source and probably acts as an antimicrobial agent because of its iron-binding capacity. It has a similar molecular weight and various other properties to serum transferrin synthesized in the liver, and it was suspected for some time that the two proteins were identical and coded for by the same gene. Conalbumin mRNA was purified by polysome immunoprecipitation and column chromatography from chicken oviduct and cDNA synthesized. This cDNA was used to demonstrate that the mRNA in liver and oviduct are identical and coded for by the same gene (Lee et al., 1978, 1980).

The chromosomal conalbumin gene is also split (Perrin et al., 1979) but it is more complex than ovalbumin, occupying 17 exons of approximately 60-200 bases long with repeated sequences within the gene and upstream from its 5' end (see below), and contains a 5' TATA box in the correct position (Cochet et al., 1979). Interesting features of the conalbumin gene are the similarity of 5' sequence motifs to the other egg white protein genes and the differences in hormone regulation between oviduct and liver; these topics are dealt with later.

Ovomucoid. Ovomucoid is a protease inhibitor. It comprises about ten percent of egg white protein. The mRNA was purified by precise sizing and affinity chromotography and the cDNA synthesized (Stein et al., 1978; Buell et al., 1979; Catterall et al., 1980). The ovomucoid gene is also split in the genome (Nordstrom et al., 1979; Stein et al., 1980) into eight exons (Gerlinger et al., 1982); the splice sites again conform to the 5' GT-AG 3' rule demonstrated for ovalbumin. The conalbumin gene appears to have evolved by two separate intragenic recombinations and the position of the introns appears to separate functional domains of the protein (Stein et al., 1980); this relationship of exons to functional domains does not hold for all proteins. A study of the processing of the primary nuclear RNA to mature mRNA showed that the introns are removed in a preferred but not an obligatory order (Tsai et al., 1980); this has also been found for many other eukaryotic genes.

One unusual aspect of gene regulation was found when transcriptional analysis was performed with the ovomucoid gene: transcription initiates at two sites, one related to a typical TATA box at around -30 base pairs, and another related to an AT rich region 85 base pairs upstream, the former being more efficient *in vivo* (Gerlinger et al., 1982). The ovomucoid gene also has three different polyadenylation sites at the 3' end and one alternative splicing possibility; all these sites function independently and give rise to multiple functional ovomucoid mRNAs, although the one with the shortest 5' and 3' end is the major species (Gerlinger et al., 1982).

Ovomucoid is a member of the kazal family of serine proteinase inhibitors. Another is ovoinhibitor, present in egg white proteins at one percent, and the gene for this protein was found just upstream from the ovomucoid gene (Scott et al., 1987). The chromosomal ovoinhibitor gene is expressed in both oviduct and liver. It is 16.3kb long and split into 16 exons. For both ovomucoid and ovoinhibitor, the introns fall between the coding sequences of the inhibitor domains; the intradomain intron occurs at an identical position in all the ovoinhibitor and ovomucoid kazal domains, suggesting that it was present in the common ancestor gene (Scott et al., 1987).

Lysozyme. Lysozyme comprises about two percent of egg white proteins. It is widely found in nature throughout vertebrates, invertebrates, plants, bacteria and viruses. It was also the first enzyme whose tertiary structure was determined (Blake et al., 1965). The lysozyme gene was cloned simultaneously from cDNA and genomic DNA libraries (Baldacci et al., 1979; Nguyen-Huu et al., 1979; Buell et al., 1979); the chromosomal gene is 3.9 kilobases long, about six times the size of the coding sequence, and it contains three introns (Baldacci et al., 1979; Nguyen-Huu et al., 1979; Lindenmaier et al., 1979). The four exons are related to the structural domains of lysozyme (Jung et al., 1980), and with the exception of exon 4, the agreement between the exons of chicken lysozyme and the structural homology with T4 lysozyme is very close (Artymuik et al., 1981).

There are multiple mRNAs for chicken lysozyme differing at the 5' end and extending to either 29, 31, or 53 nucleotides from the common initiation codon (Grez et al., 1981). There are also AT rich regions around -30 and -70 but these differ from the TATA box (Grez et al., 1981). The sequencing of the 5' end of the ovalbumin, X, Y, conalbumin, ovomucoid, and lysozyme genes permits a search for sequence homology in these steroid regulated genes; these analyses are described later.

The lysozyme gene occupies a 24kb DNase I hypersensitive chromosomal domain. At the 3' end of this domain there is a sharp transition from DNase I sensitivity to resistance, whereas the 5' transition is more gradual occurring over 6-7kb; there are eight hypersensitive sites clustered at the boundaries of the domain in oviductal DNA but only one in inactive DNA from erythrocytes (Fritton et al., 1983; Jantzen et al., 1986). There was one hypersensitive site at the 5' terminus of the active gene which maps in the region of multiple initiation sites (see above) and the putative steroid hormone control region (see below) (Fritton et al., 1983); there was no evidence of repetitive sequences associated with the boundaries of the 24kb domain (Jantzen et al., 1986).

Avidin. Avidin is a bacteriostatic biotin-binding protein also under the regulation of steroid hormones. The cDNA for chicken avidin has been cloned and has been used to confirm its hormone regulation in the oviduct, but little further structural or expression data have been reported (Gope et al., 1987). Aspects of the avidin gene, like the lysozyme gene, are highly conserved, and it shows several blocks of homology with the deduced amino acid sequence of the biotin-binding protein streptavidin from *Streptomyces* (Argarana et al., 1986).

The major egg yolk protein genes are similarly few in number. Vitellogenin, apo very low density lipoprotein II, and apolipoprotein B have all been cloned.

Vitellogenin. Vitellogenin is a large protein of 220,000 molecular weight produced in the liver of laying hens, secreted into the blood, taken up by the ovary, deposited in the oocyte, and cleaved into the two major yolk proteins, lipovitellin and phosphovitin (Redshaw and Follett, 1972; Deeley et al., 1975; Tata, 1976). cDNA for vitellogenin was initially cloned in a straightforward fashion by synthesis from purified mRNA from estradiol-treated cockerels (Deeley et al., 1977a; Burns et al., 1978; Cozens et al., 1980). A complication arose when it was realized that serum vitellogenin consisted of two clearly different proteins, VTGI and VTGII (Wang and Williams, 1980), with different responses to hormone induction (Wang and Williams, 1983), raising the possibility of more than one structural gene. Because VTGI and VTGII differed in partial proteolysis maps, it was possible to identify the cDNA for the major vitellogenin gene as VTGII (Protter et al., 1982). cDNA clones have recently been isolated for VTGI and another minor vitellogenin gene, VTGIII (Evans et al., 1988). All three genes are expressed in liver and are steroid-induced but to different levels and with different kinetics (Evans et al., 1988).

Analysis of the VTGII gene cloned from a genomic library showed its organization to be complicated, being 20.3kb in length with many introns (Wilks et al., 1981; Arnberg et al., 1981), the final tally coming to 35 of combined length 5.87kb (van het Schip et al., 1987) coding for a 1850 amino acid prepeptide. There is some conservation of number and length of introns when chicken VTGII is compared with the *Xenopus laevis* vitellogenin A2 gene (Arnberg et al., 1981).

Sequencing of the 5' end of the gene has identified a 5'-CATAAAA-3' box between nucleotides -32 and -26, and two putative polymerase binding sites around -73 and -90 nucleotides. The CR1 repetitive element, displaying structural characteristics of a retroviral long terminal repeat, has been found 2.2kb 3' to the VTGII gene and is embedded in a region of changing chromosome structure, implying a potential role in regulation (van het Schip et al., 1987).

The position of demethylation sites and nuclease hypersensitive sites within and flanking the VTGII gene have also been identified and related to hormone induction; these are discussed later.

Very low density lipoprotein II. Apo VLDL-II is also a yolk protein that is synthesized in the liver under hormonal control, and together with apolipoprotein B (see below), is the major constituent of the plasma VLDL under hormonal control (Redshaw and Follett, 1972). cDNA for chicken VLDL-II has been synthesized (Chan et al., 1976; Chan et al., 1980; Wiskocil et al., 1981) and sequenced (Wieringa et al., 1981). The 5' flanking sequence of the gene contains conserved motifs with the VTGII gene and these are discussed later. Unlike VTG, VLDL-II appears to be coded in one chromosomal gene which is split into at least four exons by at least three introns (Meijlink et al., 1981).

Apolipoprotein A-I and B. The chicken apolipoprotein A-I and B cDNA have also been cloned (Rajavashish et al., 1987; Kirchgessner et al., 1987). Whereas VLDL-II has no apparent homologue in man, the sequence and regulation of apo A-I appears similar to mammalian apo-E. The apo B gene is expressed in liver under estrogen regulation and with VLDL-II is the major component of the plasma VLDL fraction, and it is a yolk protein (Kirchgessner et al., 1987).

Regulation Of Gene Expression

Egg white protein genes. The analysis of hormonal regulation of these genes has concentrated on ovalbumin. The initial problems investigated were: whether hormonal regulation occurred at a transcriptional level or not, the kinetics and levels of induction (and tubular cell differentiation), the interaction between hormones and the extent of the coordination in induction of the four major egg white protein genes. The early analyses were performed with the use of specific antibodies, but with the availability of cDNAs these proceeded to the molecular level. Most recently interest has focused on the 5' flanking DNA sequences that are thought to be responsible for hormone induction, the interaction between the hormones and their intracellular receptors, and the binding of the hormone-receptor complex to the 5' flanking sequences of the target gene. Each of these areas will be considered in turn.

The egg white proteins are synthesized within the tubular gland cells of the oviductal magnum of the laying hen. Ovalbumin, conalbumin, ovomucoid, and lysozyme account for about 90 percent of these proteins. The magnum in an immature chicken differentiates and grows under the control of estrogen; this can be mimicked in young chicks by injections of estrogen which induces the synthesis of the four proteins. On estrogen withdrawal, protein synthesis ceases and recurs again after secondary stimulation by estrogen or progesterone, the latter being inactive for primary stimulation (O'Malley et al., 1969; Palmiter, 1972). The synthesis of the proteins is not strictly coordinated in their lag, kinetics, and extent of induction in primary and secondary stimulation (Palmiter, 1972; Palmiter et al., 1976). The early studies (Palmiter 1972, 1975) indicated that the rate of ovalbumin synthesis depends on the concentration of mRNA, and therefore hormonal control is likely to be at the transcriptional level with the single ovalbumin gene working at 35 percent of its maximal efficiency in fully-induced chickens (Palmiter, 1975), the oviductal cell mass increasing about 20,000-fold from hatch to lay.

Although there are 12,000-15,000 mRNA species in chicken oviduct and liver, in the oviduct 70 percent of the mRNA consists of about ten abundant species (Axel et al., 1976; Hynes et al., 1977). Most if not all of these species are present in both hormone-induced and withdrawn chickens but with altered frequencies (Hynes et al., 1977). The mRNAs for ovalbumin, ovomucoid, and lysozyme represent 50, 6.6 and 3.4 percent of the total poly(A)-RNA in laying hens, but only 0.01, 0.0039 and 0.0046 percent of the mRNA from withdrawn chickens, a 3000-fold difference (Hynes et al., 1977). Newly-formed mRNA_{ov} was synthesized by RNA polymerase B selectively in the chicken oviductal nuclei (Nguyen-Huu et al., 1978), suggesting transcriptional control. Using ovalbumin cDNA and filter hybridization, newly synthesized mRNA could be identified in the oviducts of induced chickens, but after 60 h of hormone withdrawal no synthesis of mRNA could be detected. After a single injection of hormone, both mRNA and high molecular weight RNA (containing intervening sequences) could be detected in nuclei after 1 h, increasing to a plateau at 8 h, results consistent with transcriptional regulation of hormone induction (Swaneck et al., 1979; Hynes et al., 1979; McKnight and Palmiter, 1979). Similar results, also indicating transcriptional control, were found for the conalbumin (Lee et al., 1978; McKnight and Palmiter, 1979), ovomucoid (Tsai et al., 1978), and lysozyme genes (Hynes et al., 1979), although with slightly different kinetics from each other and with no lag in conalbumin transcription (Hynes et al., 1979; Palmiter et al., 1976). With continuing doses of hormone (up to 14 days) the oviductal cells seem to become desensitized; although the levels of both $\mathrm{mRNA_{ov}}$ and $\mathrm{mRNA_{con}}$ remained high, protein synthesis fell into undetectable levels suggesting *trans*-acting control (Skafar and Seaver, 1985).

Estrogen is the only hormone able to induce primary oviductal cell differentiation and growth, and egg white protein synthesis, in immature chickens. It has been long known that progesterone is effective in inducing egg white protein synthesis in estrogen-withdrawn chickens (secondary stimulation) but not in untreated chickens (O'Malley et al., 1969; Palmiter, 1972), and gluococorticoid has a similar effect (Hager et al., 1980). The primary estrogen treatment may be required for the induction of other classes of steroid hormone (Hager et al., 1980). The interaction between various steroid hormones in secondary stimulation is also complex; estrogen and progesterone together have a synergistic effect on conalbumin and ovomucoid synthesis, but not on ovalbumin and lysozyme synthesis (Palmiter, 1972; Lee et al., 1978); estrogen and gluococorticoid have a synergistic effect on both ovalbumin and conalbumin induction, whereas progesterone and glucocorticoid do not (Hager et al., 1980). The role of androgen in secondary stimulation is even more puzzling; androgen cannot stimulate egg white protein synthesis on its own, but it has a synergistic effect together with estrogen, again having a disproportionate effect on conalbumin and ovomucoid synthesis (Palmiter, 1972; Tokarz et al., 1979). It was demonstrated that the androgen effect was maintained in the absence of estrogen as long as the cytoplasmic androgen receptor was present, once again suggesting a role for estrogen in induction of steroid receptors (Tokarz et al., 1979; Compere et al., 1981). This effect can be maintained up to five days after estrogen withdrawal, and does not require the presence of estrogen receptors, after which it is ineffective (Compere et al., 1981).

The conalbumin gene presents another complication in that the same gene codes for both oviduct and liver transferrin (Lee et al., 1978). Both estrogen and progesterone effected a secondary stimulation of conalbumin synthesis in the oviduct of about 96 h, whereas liver transferrin was induced slightly by estrogen (1.5 to 2-fold in 96 h) and was unaffected by progesterone (Lee et al., 1980).

There is also differential hormonal response in the ovalbumin gene and its closely linked sibling genes, X and Y. Ovalbumin is induced 10-fold higher than X or Y, and X more than Y (Colbert et al., 1980; Le Meur et al., 1981), and progesterone has a much weaker effect on X than on ovalbumin or on Y (Le Meur et al., 1981).

It is against this background of complex and complicated hormonal regulation of egg white protein genes that the molecular analysis of these interactions must take place. A further complication that must be explained is that three of the four genes are exclusively expressed in the oviduct whereas the other, conalbumin, is expressed in both liver and oviduct under differential regulation by estrogen. The promoter sequences flanking the 5' end of the ovalbumin gene were analyzed by making a hybrid construct between these sequences and part of the chick ß-globin gene as a 'reporter' gene (Knoll et al., 1983). Constructs containing differing numbers of nucleotides preceding the start of the ovalbumin gene (-753, -222, -95, -77, -48 and -24) were transfected into HeLa cells; these cells express the construct constitutively and do not respond to estrogen. The percentage expression of these constructs was 100, 100, 100, 20, 2, and 0, suggesting that the regions -77 to -48 and -48 to -24 contain regulatory sequences. In fact both these regions contain conserved sequences preceding most eukaryotic genes, the -77 to -48 region containing a sequence resembling GGTCAATCT (the CCAAT box), and the -48 to -24 region containing a TATATAT (the TATA box) (Knoll et al., 1983). The TATA box has also been found to be important in ovalbumin gene transcription by site-directed mutagenesis. The TATA sequence was converted to TGTA and the mutated sequence analyzed in a cell-free expression system. This did not interfere with correct initiation of ovalbumin RNA transcripts but markedly reduced the efficiency of gene expression (Zaruki-Schulz et al., 1982).

The 5' flanking sequences that confer tissue specificity and hormone inducibility were analyzed by transfecting the various hybrid ovalglobin constructs into primary oviductal cells and testing for regulation by progesterone. Removal of sequences from -753 to -222 still permitted progesterone induction, but the removal of sequences -222 to -95 abolished it; this identifies this latter region as being required for hormone induction, and it is in a different position to those promoter regions (-77 to -48 and -48 to -22) required for constitutive expression of the gene (Knoll et al., 1983; Dean et al., 1983, 1984).

Similar transfection experiments have identified a cell-specific regulatory element 6.1kb 5' from the start of the lysozyme gene. The element induces the lysozyme promoter in a position and orientation-independent manner and correlates with a DNase-hypersensitive site in chromatin from cells in which lysozyme is transcribed; the region contains two binding sites for the TGGCA ubiquitous nuclear DNA binding protein (Thiesen et al., 1986).

Further regulatory sequences were identified by microinjecting up to 1.4kb of lysozyme flanking sequences fused to the SV40 T antigen reporter gene. These constructs were inducible in oviductal cells by progesterone and glucocorticoid but not by estrogen, but they did not express at all in fibroblasts, and a deletion mutant lacking the sequences -161 to +15 did not express the gene (Renkawitz et al., 1982).

Lysozyme is active in macrophages as well as in oviductal cells. Chimaeric genes, with various 5' lysozyme flanking sequences fused to the HSV thymidine kinase promoter and a chloramphenicol acetyl transferase reporter gene (CAT), were transfected into chicken macrophages and fibroblasts. Two positive tissue-specific regulatory elements at around -2.7kb and -0.2kb, and a negative element at around -2.4kb that represses expression in fibroblasts but not macrophages, were identified (Steiner et al., 1987).

In another series of experiments (Dierich et al., 1987; Gaub et al., 1987), varying lengths of 5' flanking sequences of either ovalbumin or conalbumin were fused to the coding sequences of the SV40 T-antigen as a reporter gene. These were injected into the nuclei of cell lines. The ovalbumin and conalbumin promoters did not function in chicken fibroblasts, kidney cells, and a number of nonchicken cells (irrespective of the presence of steroid hormone receptors). They did, however, function in primary oviductal cells or chicken embryonic hepatocytes; the relevant sequences are near to the TATA box, between -44 and -102 for conalbumin and up to -56 for ovalbumin (Dierich et al., 1987). Chimaeric genes containing mutants in the TATA box of either ovalbumin or conalbumin had greatly reduced expression. These results are comparable with those of Knoll et al. (1983) discussed above, which located promoters for ovalbumin at -77 to -48 and -48 to -24 from the start of the gene.

The same series of chimaeric genes were tested for their ability to respond to steroid hormones (Gaub et al., 1987). A *cis*-acting negative regulatory element of ovalbumin was identified between sequences -132 and -425 which operates in the *absence* of steroid hormones; its effect is relieved in the presence of steroid hormones. This sequence will also affect the heterologous conalbumin promoter (Gaub et al., 1987). In this series of experiments no evidence was found for the positive control sequence for hormone induction reported for sequences -95 to -222 discussed above (Dean et al., 1983, 1984), although the authors themselves (Gaub et al., 1987) felt this might have been due to the limitations of their immunofluorescence assay.

It is apparent that these experiments of transfection or microinjection of chimaeric genes containing varying lengths of 5' flanking sequences are becoming difficult to interpret. It could be expected that genes from higher eukaryotes can contain several to many regulatory sequences that can interact with each other positively and negatively, and qualitatively and quantitatively, posing a complex picture of the control of gene expression. Fortunately, with the egg white protein genes one area of gene regulation, hormone inducibility, can be approached from a different angle, by purifying and studying the interaction of hormone-receptor complexes with their target DNA sequences. The chicken progesterone receptor was the first to be purified (Kuhn et al., 1975). It consists of two subunits, A and B of 79,000 and 117,000 molecular weight, with only A having a strong DNA binding capacity (Kuhn et al., 1975; Hughes et al., 1981).

It had also been known for some time (Palmiter, 1972) that the kinetics of induction of ovalbumin and conalbumin by steroid hormones was different; the accumulation of conalbumin RNA in response to progesterone is directly proportional to the nuclear progesterone receptor level, whereas ovalbumin induction requires a higher concentration of receptors, and hence levels of progesterone (Mulvihill and Palmiter, 1980); a similar effect was found when the two genes were induced by estrogen instead. $\mathrm{mRNA_{con}}$ transcription is transiently inhibited when progesterone is administered to estrogen-stimulated chickens, whereas ovalbumin gene transcription is stimulated (Palmiter et al., 1981). These observations led to the suggestion that the conalbumin gene has one hormone-receptor binding site whereas ovalbumin has several binding sites (Palmiter et al., 1981).

Partially purified chicken oviduct progesterone-receptor complex was bound to calf thymus DNA-cellulose and fragments of genomic DNA from egg-white protein genes were used to displace it (Mulvihill et al., 1982). A sequence causing strong displacement of the receptor from the column was found to reside -250 to-300 nucleotides from the start of the ovalbumin gene. There were 39 multiple homologies of this sequence adjacent to the ovalbumin X, Y, ovomucoid, conalbumin, and lysozyme genes which also strongly displaced the receptor from the column; comparison of these sequences produced the 19 base consensus: $\mathrm{ATC^{C}{}_{T}{}^{C}{}_{T}ATT^{A}{}_{T}TCTG^{G}{}_{T}TTGTA}$. Interestingly there were seven of these putative receptor binding sites 5'-flanking the ovalbumin gene and one 5' flanking the conalbumin gene (Palmiter et al., 1981).

Using a different approach, highly purified progesterone receptor (A subunit) was tested for binding to DNA fragments on nitrocellulose filters. Preferential binding was found for a 1.7kb fragment flanking the 5' end of the ovalbumin gene (Compton et al., 1982). A more detailed analysis of this region suggests that there are multiple receptor sites, the strongest of which is located between -247 and -135 nucleotides from the start of the gene (Compton et al., 1983). This result should be compared with the functional experiments performed by the same group (Dean et al., 1983, 1984), using various 5' ovalbumin sequences attached to a globin reporter gene and transfected into oviductal cells (discussed above), which identified sequences between -221 and -95 as necessary for both estrogen or progesterone-mediated transcription. Results of a DNA-cellulose competitive binding assay of the interaction of the estrogen-receptor complex with fragments of cloned DNA upstream from the ovalbumin gene identified the -140 to -21 region as binding with high affinity (Weisz et al., 1986). This overall region is also within a DNase I hypersensitive site (Kaye et al., 1986).

So far the analysis of the hormone responsive elements (HRE) of the ovalbumin gene has produced some generally consistent results, but with conflicting details. All approaches have concluded that there are one or more functional HREs flanking the gene and closely located to it, which are responsible for estrogen and progesterone (and possibly glucocorticoid and testosterone) transcriptional induction. The actual positioning of these

HRE(s) is in confusion. A more detailed analysis of the positioning and sequence of the HRE(s) requires finer analysis that would come from a detailed knowledge of the DNA (and hence amino acid) sequence of the hormone receptors themselves.

The progesterone receptor had been purified and found to consist of two subunits, A (M.W. 100,000) and B (M.W. 117,000). The B-receptor gene was cloned from oviduct cDNA libraries (Zaruki-Schulz et al., 1984; Connelly et al., 1986). The chromosomal gene was found to consist of eight exons and to be about 38kb long. It lacks TATA and CCAAT boxes, but it has CCGCCC motifs in the equivalent region (Huckaby et al., 1987). The chicken estrogen receptor cDNA was also cloned and sequenced (Krust et al., 1986) and shows highly conserved putative DNA- and hormone-binding domains compared with the human receptor. Sequence comparisons (Krust et al., 1986; Jeltsch et al., 1986) show that the progesterone, estrogen, and glucocorticoid receptors, and the c-erbA proto-oncogene, all belong to a multigene family of transcriptional regulatory proteins. This relationship between chicken steroid receptors has also been shown by the production of a monoclonal antibody, BF_4, that reacts similarly with a nonhormone-binding polypeptide of four steroid receptors, estrogen, progesterone, androgen, and glucocorticoid (Joab et al., 1984).

The cloning of the estrogen and progesterone receptors, and the conservation of their domains between receptors and across species, has allowed functions to be assigned to them. Two of these regions have been identified as domains responsible for recognition of the target gene's HREs (region C) and hormone binding (region E) (Kumar et al., 1986; Green and Chambon, 1987; Kumar et al., 1987). The binding of the progesterone and estrogen receptors have now been localized to between -58 and +1 of the start of the ovalbumin gene, occupying different but close sites (cited in Tora et al., 1988). This 59-nucleotide promoter region functions as both a progesterone and as an estrogen regulatory element in hybrid constructs, with CAT or globin as a reporter gene in chick embryo fibroblast cells (CHEF) cotransfected with chicken progesterone receptor (cPR) cDNA. Experiments cotransfecting with either or both the estrogen and progesterone receptor cDNAs showed that their HREs operated independently and additively. These experiments also demonstrated that cDNA for the larger B form of the cPR was not able to stimulate ovalbumin gene transcription in CHEF cells whereas the A form was, and that neither form was competent in HeLa cells, indicating that cell-specific factor(s) are involved (Tora et al., 1988).

The chicken lysozyme gene is also regulated by the four steroid hormones, although analysis is far less advanced than for ovalbumin. Purified progesterone receptor binds to two areas, between -200 to -160, and -80 to -50 nucleotides from the start of the gene; the glucocorticoid receptor binds to the same region with different affinities. A DNase I protection pattern on the distal site shows a longer progesterone footprint than generated by the glucocorticoid receptor (von der Ahe et al., 1986). Therefore the two receptors may bind to different but closely associated HREs.

Besides the hormone receptors, two other transcription factors, COUP and S300-II, have been identified and purified from HeLa cells and are required for the transcription of the ovalbumin genes (Sagami et al., 1986; Bagchi et al., 1987). COUP binds to a sequence GTGTCAAAGGTCAAA -90 to -70 upstream from the start of the ovalbumin gene, and binds with lower affinity to portions of the ovomucoid, Y and α-actin genes. COUP does not bind to the CCAAT promoter sequence and COUP and CCAAT binding proteins are distinct (Wang et al., 1987).

Egg yolk protein genes. The three major egg yolk proteins, vitellogenin II (VTGII), apo-very low density lipoprotein II (apo VLDL-II), and apolipoprotein B (apo VLDL-B), are all produced in the liver under estrogen induction. Most information is available for VTGII, and much less for the other two proteins. The regulation of their expression will be discussed together. Comparisons of regulation of VTGII in chickens with its regulation in *Xenopus* and *Drosphila* have also been made.

VTGII mRNA begins to accumulate in the liver 30 min after stimulation with estrogen and reaches a maximum in three days, decaying with a half-life of 30 h (Deeley et al., 1977a, 1977b). This is an increase of 750-fold from less than 0.0001 percent (10^{-5}) of the total cellular RNA to 0.075 percent. A second injection of estrogen produced an immediate response up to a peak of 0.128 percent of the RNA. The amount of VTGII protein synthesized represented nine percent and 16 percent of the liver proteins respectively (Jost et al., 1978). At 24 h after induction VTGI protein was only 1-3 percent of VTGII, but by five days had become 25 percent of VTGII (Wang and Williams, 1983).

Induction of the VLDL-II gene by estrogen produced a similar result; there was a 12,000-fold increase in RNA levels in 12 h (Chan et al., 1976, 1980). VLDL-B protein synthesis also responded to estrogen so that it was 12-15 percent of total liver protein synthesis (Capony and Williams, 1980); the synthesis of apoA-I and albumin was unaffected by estrogen (Lin and Chan, 1980). The VTGII and apo VLDL-II genes acquire estrogen responsiveness at different times during embryonic development on days 11 and nine respectively (Elbrecht et al., 1984).

The VTGII gene is heavily methylated in both estrogen-treated and nontreated birds, and estrogen induces demethylation of a site in the 5' region of the gene; the same site is unmethylated in laying hens but methylated in nonexpressing erythrocyte DNA (Wilks et al., 1982). The demethylation of this 5' site is slower than gene activation, and two other methylation sites within the gene are affected independently of DNA synthesis (Wilks et al., 1984). The VTGII gene also has several hypersensitive sites within and 3' to the gene in DNA from liver; three additional hypersensitive sites appear at the 5' end of the gene after induction with estradiol (Burch and Weintraub, 1983). Demethylation of sites at the 5' end of the gene also occurs during induction (Meijlink et al., 1983) but this occurs after the initiation of transcription and after appearance of the 5' hypersensitive sequences. The relationships of the nuclease hypersensitive sites and the demethylation to hormone regulation are discussed later.

Restriction enzyme and sequence analysis of the 5' end of the VTGII gene confirmed that the undermethylation site is situated at -611 nucleotides from the cap sequence flanked by a region rich in A+T sequences (Geiser et al., 1983). A TATA box (5'-CATAAAA-3') was also found between nucleotide -32 and -26, and two sites analogous to the bacterial RNA polymerase-binding site, between -77 and -69, and between -101 and -90 (Geiser et al., 1983). Purified estrogen-receptor complex was bound to chicken DNA on cellulose and to cloned fragments of the VTGII gene in a displacement assay (Jost et al., 1984). The fragment that gave the highest competition was -458 to -725, which contains the estrogen-dependent hypomethylation site. *In vitro* methylation of this site does not affect competition, whereas cleavage of it causes complete loss of competition. These results and those of DNase I protection experiments suggest the probable site is the 24-nucleotide 5'-GCGTGACCGGAGCTGAAAGAACAC-3' (Jost et al., 1984). The eight 5' nucleotides of this sequence are *part* of the conserved sequence in four *Xenopus* genes and the chicken apoVLDL-II gene (5'-GGTCANNNTGACC-3'), but the rest of the sequence finds no homology.

A functional assay for the role of the sequence around -611 was investigated using an *in vitro* chicken embryonic liver nuclei system (Jost et al., 1985). An avian VTGIIcauliflower mosaic virus hybrid gene was made containing 1600 nucleotides of the 5' flanking sequences. In this system the gene was transcribed and induced by the addition of purified estrogen-receptor complex (the endogenous VTGII gene in the embryonic nuclei remained inert); deletion or inversion of the estrogen-receptor binding site (-620 to -597) prevents induction (Jost et al., 1985).

The 5' flanking 2030 nucleotides of the VTGII gene and 2594 nucleotides of the VLDL-II gene were sequenced (van het Schip et al., 1986). Sequences with limited homology to the -621 to -597 VTG sequence were found in apo VLDL-II at -1629 and -1452. In both genes a sequence resembling the glucocorticoid responsive element (GRE) is overlapping and downstream of these sites (van het Schip et al., 1986). A further-conserved 20-nucleotide block of homology is found in the -100 to -300 region of both genes, but in different orientation, and in the four *Xenopus* VTG genes; it is not found in egg white protein genes and its role is unknown (van het Schip et al., 1986).

By comparing the 5' flanking sequence of four *Xenopus* VTG genes, four blocks of homology were found between -400 and the start of the genes; these same blocks are also found in chicken VTGII (Walker et al., 1984). One block, a palindromic sequence 5'-GGTCANNNTGACC-3', is conserved in position (around -340) in all five genes and also in two copies close to the 5' end of the chicken apo-VLDL-II gene (Walker et al., 1984). When various constructs were made between the 5' flanking sequence of the *Xenopus* B1 VTG gene and the CAT reporter gene and transfected into human MCF-7 cells, it was found that this 13-nucleotide element (ERE; 5'-AGTCACTGTGACC-3') was essential for estrogen inducibility (Seiler-Tuyns et al., 1986). Similarly a 35-nucleotide sequence from the 5' flanking region of the *Xenopus* A2 VTG gene, containing the 5'-GGTCACAGTGACC-3' palindromic sequence, was able to confer estrogen responsiveness on the thymidine kinase promoter and express CAT in the MCF-7 cells (Klein-Hitpass et al., 1986). This indicates that the ERE has been conserved in evolution and operates in a human cell line with a heterologous promoter.

The 13-nucleotide ERE identified in the VTG and VLDL-II genes is very similar to the GRE consensus sequence of 15 nucleotides. The GRE is recognized by the progesterone receptor, and the ERE element can be converted into a GRE by the substitution of one or two bases at homologous positions in the palindrone (Klock et al., 1987). These elements therefore form part of a related family of DNA sequences.

The analysis of the 5' flanking sequences of chicken VTGII and VLDL-II has identified two sequences associated with estrogen induction by different criteria, a 24-nucleotide sequence from -621 to -597 associated with estrogen-induced hypomethylation and DNase I sensitivity and which when deleted prevents induction of a hybrid gene in embryonic nuclei, and a 13-nucleotide palindrone conserved in VTGII, VLDL-II, and from *Xenopus* VTG genes and which confers estrogen-induction on a heterologous promoter in a human cell line.

It has also been shown that the VTGII gene sequences are associated with the nuclear matrix of chicken liver, but they are not in the oviduct of laying hens, and this includes 2kb 5' and 2kb 3' flanking the gene. Fifteen days after estrogen induction the gene is no longer preferentially associated with the nuclear matrix (Jost and Seldran, 1984).

MUSCLE PROTEIN GENES

Muscle proteins have been intensively studied because myogenesis in embryos goes through clearly defined developmental stages from myoblasts to myofibrils. Chickens have often been used for this work because embryonic material of the correct stage is readily available as are large quantities of adult muscle as the starting point for gene cloning.

The myoblasts contain about 17,000 different RNA species in three or four different abundance classes. After differentiation into myofibrils, however, there are about 2,500 sequences of higher concentration, with six sequences of exceptionally high concentration of about 15,000 copies per nucleus each (Paterson and Bishop, 1977). These six abundant sequences no doubt represent the common adult muscle protein of actin, myosin, tropomyosin, etc., and the molecular genetics of each of these will be discussed in turn.

Actin genes. There are between six and 11 isoforms of actin that have been identified biochemically in chicken tissues. Recently six different chicken actin genes have been cloned (Chang et al., 1984); partial sequencing of these genes allowed their identification when compared with protein sequence information. They included the genes for α-skeletal, α-cardiac, and α-smooth muscle actin. They also included nonmuscle genes ß-cytoplasmic actin, an unidentified actin, and a previously unknown actin similar to the amphibian type 5 actin (Chang et al., 1984). Genomic blots with these cloned sequences showed that the actin multigene family consists of 8-10 loci and therefore each actin isoform seems to be coded for by a different gene.

The nucleotide sequence of the α-cardiac actin gene showed that it contained a promoter and six introns (Eldridge et al., 1985). There are no strong homologies between the promoter and 3' untranslated regions of the α-skeletal and α-cardiac actin genes. In myoblasts the ß and γ nonmuscle actin genes are expressed and the α-skeletal and α-cardiac actin genes are sequentially induced on fusion to form myotubes.

Specific ß-cytoplasmic, α-skeletal and α-cardiac probes showed that at day 10 *in ovo* embryonic hindlimbs contained low levels of actin mRNA consisting mainly of ß and α-cardiac types. By day 17 *in ovo* the amount of α-skeletal had increased 30-fold to about 90 percent of the actin RNA, whereas the levels of α and ß-cardiac had decreased by 30 and 70 percent respectively (Hayward and Schwartz, 1986). In proliferating myoblasts in culture, ß-actin mRNA increased sharply and steadily declined at fusion; α-cardiac increased to 15-fold higher than α-skeletal in profusion myoblasts (36 h) and remained high. α-Skeletal actin mRNA, however, remained low until fusion had begun (48 h), and increased 25-fold by the end of fusion, and then decreased. Therefore the events *in vitro* resemble those *in ovo*, except for maintainence of high levels of α-cardiac and the drop in α-skeletal after fusion.

Using fusions of different portions of the 5' flanking sequences of chicken α-cardiac actin gene to CAT as a reporter gene, it could be shown that a sequence within 202bp (Bergsma et al., 1986) from the start site which was downstream from the CCAAT sequence (Quitsche et al., 1988) was responsible for tissue-specific expression in transfected myotubes; the precise sequence of the CCAAT box had no effect in itself. The ß-actin promoter was constitutive, equally active in myoblasts and myotubes, and dependent on a direction-specific sequence upstream of its CCAAT box (Quitsche et al., 1988). But if 3' flanking sequences were included in the construct, down-regulation of ß-actin mRNA occurred in contractile myotubes (Arnold et al., 1988).

5' flanking sequences for both α-skeletal and ß-cytoplasmic actin genes, as well as the histone H2b gene were fused to a CAT reporter gene and transfected into myogenic and nonmyogenic cells (Grichnik et al., 1986). The expression of the α-skeletal actin-CAT construct was restricted to myogenic cells and was induced nine to 15-fold during myoblast fusion; a 411-nucleotide fragment, 5' to the start of the gene, was responsible for the cell type and stage specific expression (Grichnik et al., 1986). With a similar series of experiments the sequence controlling expression was located within 200 nucleotides 5' to the start of the gene (Bergsma et al., 1986). Microinjection of the α-actin-CAT constructs into *Xenopus* oocytes defined a *cis*-acting transcriptional control region with 107 nucleotides of the gene; the CCAAT and TATA box homologies lie within this region.

The structure and sequence of the α-smooth actin gene has also been determined (Carroll et al., 1986). It contains eight introns, and unlike other actin genes it produces multiple mRNAs; there are at least four species from 1370-2700 nucleotides long differing in the length of their 3' untranslated region. A similar series of 5' flanking region constructs were made between α-smooth muscle actin and the CAT reporter gene and transfected into fibroblasts (with high levels of α-smooth muscle actin expression) and myoblasts (which accumulate lower amounts of mRNA). The first 122 nucleotides 5' to the gene contained a promoter capable of controlling high levels of transcription in both fibroblasts and myotubes (Carroll et al., 1988). Another regulatory element was located between -257 and -123, which modulated the level of expression in fibroblasts, and contained an evolutionarily conserved sequence and one of the pair of 16-nucleotide inverted CCAAT box-associated repeats found among all chordate muscle actin genes. A smaller segment, -151 to -123, of these repeats was sufficient to suppress expression from the promoter in muscle cultures, suggesting that the upstream CCAAT box repeat plays a negative role in α-smooth muscle actin gene expression.

Myosin genes. As with actin, a large number of isoforms of vertebrate myosin have been identified that are cell and stage specific. Each myosin unit consists of two heavy chains (MHC; Mr 210,000) and two to four light chains (MLC; Mr 17,000-21,000).

Myosin heavy chains. In chicken skeletal muscle, three MHC isoforms each with a unique peptide map appear sequentially in development (Bandman et al., 1982). An embryonic isoform is expressed early and throughout development in the embryo; it is replaced by a second form after hatching which predominates by ten days; the third adult isoform predominates by eight weeks of age. *In vitro*, muscle cultures express only the embryonic form even after the transition from myoblast to myotubes. The MLC fast and slow isoforms are expressed in embryos until day 14 when there is an increase in MLC fast chain 3 accumulation; this shift does not occur with differentiation *in vitro* (Bandman et al., 1982).

The mRNAs for fast and slow MHC were purified by immunoprecipitation of the polysomes from embryonic chicken skeletal muscle used to screen cDNA clones from the same tissue, and two fast MHC genes were identified (Umeda et al., 1981). Similarly prepared cDNA probes were used to screen a genomic DNA library (Robbins et al., 1982), and 11 positive clones were identified which fell into seven distinct groups by restriction enzyme analysis. All these clones code for muscle-specific MHC isoforms, and there are at least three size classes of mRNA sequences whose relative amounts are tissue and stage specific (Robbins et al., 1982).

Two of these genes code for fast-white MHC isoforms; one is expressed in the embryo, the other in adult muscle (Gulick et al., 1985). The 5' untranslated region of the adult gene contains one intron and the embryonic gene contains two. Both genes contain the conserved CCAAT and TATA boxes although these were the only homologies in the 5' flanking sequences. The structural sequences at the 5' end of the two genes were highly conserved, as were the position of their first three introns (Gulick et al., 1985).

A fragment of the adult gene, containing 1400 nucleotides of 5' flanking sequence and 1600 bases downstream from the initiation codon, was used to screen two genomic chicken libraries (Robbins et al., 1986). Thirty-one unique nonoverlapping clones were isolated, and all showed homologies in the coding region but with no extensive homologies in the 5' flanking region. Seven of these genes fell into a group with almost identical DNA sequences for the ATP binding site domain (Robbins et al., 1986) and these included the adult and the embryonic genes previously analyzed (Gulick et al., 1985). The sequences in the 5' flanking region of the genes (Kropp et al., 1986) showed that the genes have extensively diverged; the first and third introns have also diverged, but the second intron is conserved and includes a 24-nucleotide repeated sequence restricted to MHC genes (Kropp et al., 1986). The sequence of the three exons at the 5' end are highly conserved and all the genes contain CCAAT and TATA boxes at the expected locations (Kropp et al., 1987). Although there are short conserved sequences in the 5' flanking regions of the genes, it is not possible to group them into any pattern relating to a particular gene's function. Only one sequence was found that was muscle-specific and found in other genes (for instance in actin, troponin I, troponin T, MLC; see later) and that was the CATTCCT sequence located at varying positions from the start of the gene (Kropp et al., 1987). For six of the seven genes, a transcript-specific probe could be made and each produced a unique pattern of expression in relationship to muscle type and stage of development (Kropp et al., 1987).

One of the embryonic MHC genes has been completely sequenced (Molina et al., 1987). The complete gene encompasses 23kb and has 37 introns, and it reveals evolutionary conservation in those regions of the protein which bind ATPase and actin.

Myosin light chains. Myosin light chains (MLC) are the alkali and regulatory components of myosin molecules which are organized into the large filaments of myofibrils. Multiple isoforms exist in both different types of muscle and at different stages in development. The genes for several of these proteins have now been cloned.

The first gene isolated was for an embryonic cardiac MLC polypeptide (Arnold and Siddiqui, 1979). This 613-nucleotide cDNA (Arnold et al., 1983) was used to clone the chromosomal gene from a genomic DNA library (Winter et al., 1985). This gene (MLC-2A; LC_{2A} polypeptide) extends over 4.2kb, it is interrupted by five introns, and it is present in a single copy in the genome. In agreement with its mode of expression, the MLC-2A has heart-specific DNase I hypersensitive sites (Winter and Arnold, 1987). MLC-2A 5' flanking sequences were fused to CAT as a reporter gene and transfected into various primary cultures. Only 150bp of 5' flanking upstream of the start site was sufficient for high levels of expression in both embryonic chicken breast and heart muscle cells but not in fibroblasts (Arnold et al., 1988); this tissue-specific expression was suppressed in breast muscle by BUdR, a reversible inhibitor of myogenic differentiation.

cDNAs have also been isolated for MLCs from embryo (LC_1) and adult (LC_3) leg skeletal muscle (Nabeshima et al., 1982). The C-terminal 141 amino acids and the 3' untranslated regions were identical, but the two cDNAs differed in the 5' untranslated region and the N terminal 35 amino acids of LC_1 and 8 amino acids of LC_3 (Nabeshima et al., 1982). Once a genomic DNA library was screened it was found that both cDNAs were encoded in a chromosomal gene of 18kb (Nabeshima et al., 1984). Two precursor RNAs of 17.5 and 8kb are transcribed and processed by differential splicing to form mRNAs encoding the two distinct light chain proteins, LC_1 and LC_3, involving shuffling of the four 5' exons out of the nine (Nabeshima et al., 1984). This differential splicing allows developmental regulation whereby LC_1 synthesis precedes LC_3 synthesis during myogenesis. About 1kb of DNA 5' from the start of the MLC 1/3 gene was sufficient for differentiation-dependent tissue-restricted expression when transfected into primary chicken myoblast cultures using CAT as a reporter gene (Billeter et al., 1988). The expression of CAT activity from both promoters was ten to 20-fold higher in differentiated myotubes than in fibroblasts or myoblasts, although the relative timing of LC_1 and LC_3 expression was altered (Billeter et al., 1988).

The LC_1 cDNA clone was used to isolate two further cDNA clones for MLCs from adult gizzard and fibroblast cDNA libraries (Nabeshima et al., 1987). These two cDNAs were not identical to the MLC 1/3 gene although they were part of the same MLC family and showed strong identity with each other. The gizzard and fibroblast cDNAs were identical in coding sequence except for a 26-nucleotide block (nine C-terminal amino acids) addition in fibroblast cDNA; they are also identical in both 5' and 3' noncoding sequences. Because there was only one chromosomal gene, it was suggested that the two cDNAs were generated by a further exon shuffling mechanism (Nabeshima et al., 1987).

The LC_1 cDNA was also used to screen an embryonic gizzard muscle cDNA library, and a cDNA for MLC L_{23} was identified (Kawashima et al., 1987). This cDNA was also found to be a member of the alkali type MLC gene family, but it was a distinct gene. The gene is expressed transiently in embryonic skeletal, cardiac, and smooth muscles and is also found in the brain at all stages of development.

Tropomyosin genes. These proteins were originally identified in skeletal muscle but are also found in nonmuscle tissue. In muscle they are components of the actin-containing thin filaments, and in nonmuscle cells they are associated with the actin-containing microfilaments of the cytoskeleton. In chicken skeletal muscle there are two major forms, α and ß, which can be separated on PAGE, as well as variants of α found in different muscle types. cDNAs have been synthesized for two distinct mRNA species of α-tropomyosin from chicken leg muscle (MacLeod, 1982). These varied considerably in coding sequence, the minor leg muscle form being similar to that found in slow twitch muscle.

In the smooth muscle of the gizzard of 17-day chicken embryos there are three tropomyosin polypeptides, α,ß and γ, each with a major and a minor form. Separation of the mRNAs for these three species suggested that they were coded for by separate genes (Segura and Saborio, 1982). cDNA from stomach and gizzard smooth muscle of 11-day-old embryos was isolated from a DNA expression library (Helfman et al., 1983). One plasmid contained the entire coding region and some 5' and 3'-untranslated regions (Helfman et al., 1986). This smooth muscle α-tropomysin cDNA showed extensive homology with skeletal muscle sequences from chicken (α) and rabbit (α and ß) and a horse platelet ß-sequence. The greatest divergence from skeletal muscle sequences is in the COOH-terminal region.

Troponin genes. The troponins I, C and T form a complex with tropomyosin and are involved in calcium ion binding and release in muscle contraction. As with other contractile protein genes, the troponins are members of a multigene family with diverse protein isoforms developmentally-regulated and tissue-specific.

A troponin T (Tn-T) cDNA was isolated from chick embryonic skeletal muscle (Ordahl et al., 1980). This turned out to be the cardiac Tn-T gene which was transiently expressed in embryonic skeletal muscle (Cooper and Ordahl, 1984). This abrupt repression of Tn-T expression is regulated at the transcriptional level (Long and Ordahl, 1988). The cardiac Tn-T gene has 18 exons and also produces different embryonic heart and adult heart protein isoforms by exon shuffling, with a single promoter being used for both skeletal and cardiac expression patterns (Cooper and Ordahl, 1985). The rat Tn-T gene is closely conserved to the chicken gene but with an even greater capacity for differential splicing (Breitbart and Nadal-Ginard, 1986).

The chromosomal gene for the fast skeletal muscle form of chick troponin-I (sTn-I) has been cloned (Nikovits et al., 1986). It is 4.5kb long with eight exons and codes for a major and a minor mRNA species, although there is only one protein species in both embryonic and adult skeletal muscle. A comparison of the sequences 5'-flanking 15 mammalian and avian muscle genes identified a conserved consensus sequence: 5'-CATTCCT-3'.

Analysis of troponin-C (Tn-C) expression in cardiac muscle indicated its developmental profile to be different to actin or myosin (Malhotra and Bag, 1987). Detailed molecular analysis of Tn-C expression will have to await cloning of the gene.

Other contractile protein genes. Creatine kinase is a dimer, and in myoblasts and embryonic cells the BB isoenzyme predominates being replaced by the adult muscle-specific M subunit on terminal differentiation. cDNA for CK-M mRNA has been synthesized (Rosenberg et al., 1982; Schweinfest et al., 1982). It hybridizes to RNA from differentiated muscle but not to that from myoblasts, smooth muscle or nonmuscle cells.

The levels of the glycolytic enzyme, glyceraldehyde 3-phosphate dehydrogenase (GAPDH), are also induced during muscle cell differentiation, although it is expressed in myoblasts at low levels. The gene has been cloned and partially sequenced from chicken embryonic heart (Arnold et al., 1982), breast muscle from 3-week-old chicks (Dugaiczyk et al., 1983), and embryonic brain (Milner et al., 1983). The chromosomal gene is 4.6kb long and contains 12 exons; it has a TATA box with extremely G+C rich 5' flanking sequences including two 9-base-pair inverted sequences (Stone et al., 1985). The levels of both GAPDH mRNA and protein are the same in embryonic brain, liver, and muscle, whereas they are elevated in adult muscle and lowered in brain and liver, suggesting regulation at the transcriptional level (Milner et al., 1983). Adenylate kinase (AK) is an enzyme involved in adenine nucleotide homeostasis, and its expression has a similar developmental and tissue-specific profile to GAPDH. cDNA for AK has been isolated from a chicken skeletal muscle cDNA library, but other than showing the induction of muscle AK mRNA during embryogenesis (Kishi et al., 1986) no further details have been reported.

α-Actinin is a component of the Z-line of skeletal muscle and of similar structures in smooth muscle and the cytoskeleton of nonmuscle cells. A complete α-actinin cDNA has been cloned from a chicken fibroblast λgt 11 library (Baron et al., 1987). It encodes the smooth muscle form of the protein. Although a considerable number of isoforms of α-actinin exist in a tissue, the sequence of ten different peptides from chicken smooth muscle was found to reside in this cDNA, and no hybridizing RNA species could be found in skeletal muscle, suggesting that skeletal α-actinin is encoded in a different divergent gene (Baron et al., 1987); α-actinin contains homologies in part to both spectrin and calmodulin.

GLOBIN GENES

Like mammalian globins, those of chickens are coded for by two gene families, α-like and ß-like, situated on different chromosomes (Hughes et al., 1979).

α-globins. The adult α^A and α^D and embryonic globin genes were isolated from a chromosomal DNA library (Engel and Dodgson, 1980). The two highly divergent adult α genes were separated by 2.4kb and contained two introns of similar size to the mouse globin gene. The embryonic α-globin gene (π') was also found in this linkage group, at the 5' end followed by the adult minor α^D and then the adult major α^A (Dodgson et al., 1981); an earlier identification of an anemic shock-induced specific α globin, α^S, was found to be α^A. The embryonic α-globin gene, π', has an unusual structure in having substantially larger introns similar to the human embryonic α-globin gene ζ (Engel et al., 1983). A further embryonic α-globin peptide, π, was found to be allelic to π' (Engel et al., 1983).

The chicken α-globin genes are all transcribed in the same direction, 5' -π' -α^D -α^A -3'. Their sequence (Dodgson and Engel, 1983) shows that, unlike human adult α-globin genes, all three are widely divergent and do not cross-hybridize. Within this 30kb chromosome domain there are repetitive DNA sequences that map within, or adjacent to, AT-rich DNA domains, and frame the domain correlating with clusters of sites of DNase I sensitivity and differential methylation, and sites of DNA matrix attachment (Broders et al., 1986). Indeed there is evidence that the whole domain produces heterogeneous large transcripts of 12kb or more, containing the two adult and the embryonic globin gene (Broders and Scherrer, 1987).

ß-globins. An adult and an embryonic ß-like globin gene were cloned from a chromosomal DNA library (Dodgson et al., 1979). The adult gene was 5' to the embryonic gene and contained one large and one smaller intron, analogous to mammalian ß-globin-like genes; the embryonic gene contained at least the larger intron. It was later found that the total ß-like family of genes was a cluster with two embryonic genes, ε and ρ, and two adult genes, the major and the minor, in the order 5' -ρ -$ß^H$ -ß -ε -3'; the embryonic genes are at each end of the cluster, an arrangement unique in vertebrates (Dolan et al., 1981).

The nucleotide sequence of the adult major ß-globin gene ß or $ß^A$ (Dolan et al., 1983) shows a structure analogous to mammalian globin genes, with introns of 92 and 810bp. There are also CAAT and TATA boxes at -74 and -30 nucleotides from the cap site (Dodgson et al., 1983). The nucleotide sequence of the embryonic ß-like globin genes, ρ and ε (Dodgson et al., 1983), show a structure typical of functional ß-like genes with small 5' introns and larger 3' ones. Both genes have consensus flanking sequences similar to the adult ß gene (Dodgson et al., 1983; Dolan et al., 1983) and are similar to each other from -130 nucleotides from the cap site and within intron I; intron II and sequences 5' to -130 are highly divergent (Dolan et al., 1983). The embryonic genes are more similar to the adult ß-globin gene than are the mammalian ones, suggesting a separate evolutionary pathway (Dolan et al., 1983).

A DNase I hypersensitive site was found between about -260 and -60bp from the cap site which was correlated with gene activity; it is present in erythrocytes of 14-day embryos but not in primitive erythrocytes of 5-day embryos (McGhee et al., 1981). This 200bp domain was also identified by footprinting (Jackson and Felsenfeld, 1985), the first application of this technique. This led to the partial purification of specific nuclear factors that bound to the nuclease-hypersensitive domain (Emerson et al., 1985). Two discrete protected regions within this domain were identified, each appearing to bind one or more distinct components, acting independently, the complexes being present in the transcriptionally active $ß^A$ gene (Emerson et al., 1985). These DNA-binding proteins were fractionated by HPLC (Plumb et al., 1986). Three $ß^A$-globin binding activities were separated, each binding a different 5' site, designated A, B, and B'. An erythroid precursor cell line has sites B and B', but binding to site A is only detected after the cells begin to differentiate (Plumb et al., 1986); the B' site contains the globin concensus sequence CACC. The minor adult $ß^H$-globin gene has two 5' flanking DNase I hypersensitive sites which bracket two binding sequences H and H'. The H and B sites contain variants of the sequences bound by Nuclear Protein-I and the TGGCA-binding protein (Plumb et al., 1986). It has been also shown that histone octamers show a strong preference to be positioned over -206 to -62bp before the cap site, the region which contains the sequences identified as binding specific protein factor and containing the promoter sequence motifs, indicating precise nucleosome placement (Kefalas et al., 1988).

A series of hybrid constructs between different portions of the $ß^A$ gene and the CAT gene were used to investigate regulation of expression in embryonic erythrocytes (Hesse et al., 1986). It was shown that promoter activity of the $ß^A$ 5' flanking sequence was strongly stimulated by a sequence 110-588bp 3' to the poly(A) signal (Hesse et al., 1986). The effective 5' promoter region is within 112bp of the cap site, and the 3' enhancer is within a 300bp Pvu II fragment lying about 400bp 3' to the poly(A) signal and controlling cell specificity and timing of induction of ß-globin transcription (Choi and Engel, 1986); a similar sequence is found in ducks (Kretsovali et al., 1987). Detailed deletion analysis and transient expression analysis have shown this 3' enhancer to be about 136bp long, and specific factors are found in adult erythrocytes that bind to it (Emerson et al., 1987). These factors are specific to tissue or developmental stage and they bind to four discrete regions within the enhancer and one near it; this also corresponds to a nuclease hypersensitive site. The appearance of these factors during development suggests they may be responsible for the specificity of enhancer activity (Emerson et al., 1987).

CALMODULIN GENES

Calmodulin (CaM) is an intracellular calcium receptor that exists in all eukaryotic cells and is evolutionarily highly conserved. A short cDNA for chicken CaM was isolated from partially purified mRNA (Munjaal et al., 1981) and used to isolate a full-length eel CaM, which in its turn was used to isolate cDNAs containing the entire chicken coding sequence (Putkey et al., 1983). The overall nucleotide homology of chicken and eel coding regions was 79 percent but the 5' and 3' nontranslated sequences are much diverged (Putkey et al., 1983). When a chicken chromosomal cDNA library was screened (Putkey et al., 1983; Stein et al., 1983), two genes were identified, cCL1 and cCM1.

CL1 produces two mRNAs of 1600 and 1900 nucleotides in the ratio of 4:1 in all tissues by differential splicing. The gene is 12kb long with eight exons, interrupted by introns varying in size from 19bp to about 4350bp (Simmen et al., 1985). A TATA box was located at -29 and a CCAAT sequence at -58bp from the cap site. The genomic organization is highly conserved compared with other calcium binding proteins.

The second gene, CM1, is most unusual (Stein et al., 1983) because it does not contain introns, which at first suggested that it originated as a processed gene copy of CL1. Unlike other processed genes, CM1 is functional and its expression is specific to muscle cells; it has 19 amino acid substitutions compared with CL1. Synthesized CM1 protein shows, however, very similar properties to ordinary calmodulin (Putkey et al., 1985). Once the muscle-specific cDNA was isolated, however, the CM1 gene was found to encode slow-muscle troponin C and not to be a product of a pseudogene (Putkey et al., 1987). The hybridization between CM1 and troponin C cDNA is due to a short region of high homology and both genes exist as distinct single copies.

This similarity is hardly surprising in that calcium homeostatis is regulated by a broad family of binding proteins including calmodulin, troponin C, parvalbumin, oncomodulin, and vitamin D-dependent binding protein (CaBP-28). A cDNA for chicken CaBP-28 was isolated from a λgt11 library together with another cDNA which had 84 percent nucleotide and 99 percent amino acid homology to chicken brain calmodulin; this latter cDNA was termed neo-calmodulin (neo CaM). Both neo CaM and CaBP-28 cDNAs each recognize three mutually exclusive mRNA transcripts, the CaBP-28 transcripts being induced by vitamin D (Mangelsdorf et al., 1987).

COLLAGEN

The collagens are a large family of structural proteins in the extracellular matrix of eukaryotes. Type I is the most common being found in bone, tendons, and skin; type II is the main collagen of cartilage being secreted by the chondrocytes; type III is found in blood vessels, uterus, and fetal skin; types IV and V are in basement membranes. Types I, II, III, and V form characteristic fibrils and have a structural role, whereas IV, VI, VII, VIII, IX and X collagens do not and are short-chain. It is clear from the protein characterization that the collagens must be a diverged multigene family.

The α^2 type I collagen gene was cloned from a genomic DNA library and found to be at least 30-40kb long and contain more than 50 exons (Ohkubo et al., 1980; Wozney et al., 1981); these exons appear to be an amplification of a 54bp exon (Yamada et al., 1980; Dickson et al., 1981). The 5' flanking sequences of the gene contain a TATA box at -33 to -26 and a CCAAT box at -84 to -78 (Vogeli et al., 1981). Both the mouse and the chicken 5' flanking sequences are sensitive to S_1 nuclease digestion; in the chicken the sequence is -80 to -200bp before the cap site (McKeon et al., 1984). The α^1 type I collagen gene has also been cloned and the cDNA sequenced (Fuller and Boedtker, 1981). Type I collagen is the most abundant type and is composed of two α1 chains and one α2 chain. The nucleotide sequence has 84 percent homology with a 49bp region surrounding the translation start point of the α2-I gene (see below) and 70 percent homology in the nucleotide sequences encoding the N-propeptide triple helical region of the two type I collagen chains (Finer et al., 1987b). The 5' flanking sequence of the α1-I gene contains both TATA and CCAAT boxes but it is missing the 40-nucleotide pyrimidine stretch around -165, which corresponds to the S1-nuclease hypersensitive site and which is present in other collagen genes (Finer et al., 1987a). There are two other unusual sequences in the α1-I gene; first, there are fifteen tandem repeats of the sequence GGGGAGA within the first intron (correlated with S1-nuclease hypersensitivity), and second, there are at least 25 copies of a polymorphic 23bp tandemly repeated sequence not found in other type I genes; both these repeated sequences are found elsewhere in the genome (Finer et al., 1987a).

cDNA for α1 type II collagen was prepared from chick sternal cartilage (Vuorio et al., 1982) and used to screen a genomic DNA library and 6kb at the 3' end of the gene isolated (Sandell et al., 1983). There is only one α1 type II gene; the cartilage 3α collagen chain, which is very similar to the α1 II chain, must be due to differential processing (Sheffield and Upholt, 1985). So far 46 exons for the α1 type II gene have been identified; this gene differs from other collagens in having introns of considerably smaller size down to a minimum size of about 80bp (Upholt and Sandell, 1986).

The α1 type III gene has also been isolated (Yamada et al., 1983b) and is present in crop mRNA but not calvaria mRNA as would be predicted; there are at least 16 exons in a 9kb DNA segment.

The sequence base composition and codon usage of the α2 type I gene is very similar to that of the α1 type I and differs from those of the α2 type III gene (Sandell et al., 1984). The deduced amino acid sequence for the C propeptide has strong homology to the equivalent C propeptide of α1 and α2 type II proteins; the organization of the terminal four exons is the same, predating gene duplication (Yamada et al., 1983a).

In chicken embryonic development, the production of collagen-enriched tissue occurs between days 7 and 15. There is a coordinate ten-fold induction of mRNAs for the α1-I, α2-I, α1-II, and α1-III genes between days 5 and 10; this induction is specific to collagen genes (Merlino et al., 1983). The α2-I gene has DNase I hypersensitive sites at the 5' end at both day 2 and 5 of embryonic development, and the same gene is expressed in early and late embryos (Merlino et al., 1983). The α1-II mRNA increases from 20 copies per diploid genome to 2,000 copies during limb cartilage differentiation, and up to 10,000 copies in sternal cartilage (Kravis and Upholt, 1985).

Several newly discovered tissue-specific 'short-chain' collagen genes have also been cloned in chickens. Type X collagen is found in hypertrophic chondrocytes localized to the mineralization zones of hyaline cartilage; it is therefore transient and developmentally regulated. Both cDNA and genomic clones of the type X gene have been isolated and contain a segment with two features of a transmembrane domain (Ninomiya et al., 1986). The genes for the α1-IX and α2-IX collagens have also been cloned (Ninomiya et al., 1985); the collagens are found in hyaline cartilage and are shorter than the α-chains of types I, II and III and are heterotrimers of three genetically distinct subunits containing three triple-helical collagenous domains interspersed with noncollagenous domains (Ninomiya and Olsen, 1984; Ninomiya et al., 1985). These type-IX collagen genes were used to isolate another novel collagen gene from a tendon fibroblast cDNA library (Gordon et al., 1987). This new type-XII gene is expressed in several tissues of chicken embryos and is part of the type-IX multigene family distinct from the fibrillar collagens (Gordon et al., 1987).

CRYSTALLINS

The α, β, γ and δ crystallins are about 80 percent of the soluble proteins of the lens of the eye. δ-crystallin is present only in birds and reptiles and has been analyzed in most detail in chickens.

δ-crystallin is encoded by two genes; cDNA for the δ1 crystallin was cloned and sequenced from an embryonic lens library (Nickerson and Piatigorsky, 1984; Yasuda et al., 1984). These cDNA clones were used to isolate both the δ1 and δ2 genes from genomic DNA libraries; the genes are about 4.2kb apart orientated with the same polarity 5'-δ1-δ2-3', and both have 17 exons and 16 introns (Hawkins et al., 1984; Ohno et al., 1985). Both genes have been sequenced. The δ1 gene is 7.76kb long with a suggestion of a 2-fold repeat in protein structure (Nickerson et al., 1985; Ohno et al., 1985). The δ2 gene has exons 3-17 identical in size to δ1 with sequence homologies from 70-100 percent, a protein homology of 91 percent, with a TATA box and consensus splice junction. Although only δ1 is known to be expressed, it is possible that δ2 is a functional gene (Nickerson et al., 1986). To analyze expression further, the 5' flanking regions of the δ1 and δ2 genes were made into hybrids with the CAT gene as a marker, and transfected into embryonic lens epithelial cells (Borras et al., 1985). The δ1 promoter region is much stronger and contains a CCAAT sequence at -71 and two viral core enhancer sequences absent from the δ2 gene (Borras et al., 1985). The two δ crystallin polypeptides found in the chicken lens are produced from the δ1 gene. No δ2 cDNA can be isolated from the lens (Wawrousek et al., 1986). When different portions of the 5' δ1 gene flanking sequences were incorporated in the CAT constructs, a sequence between -120 and -43 was found to have strong promoter activity, which was reduced 20-fold when a sequence -603 to -120 was included in the construct (Borras et al., 1988). The -120 to -43 region is G+C rich, operates in HeLa cells (and so is not lens-specific), and binds an Sp-1-like transcription factor from HeLa cell extracts (Das and Piatigorsky, 1986).

Similar experiments using a CAT reporter gene have also demonstrated that the region upstream of -100 is not necessary for lens-specific expression (Hayashi et al., 1985). A segment spanning the third intron was identified as having strong lens-specific enhancer activity (Hayashi et al., 1987). This enhancer increases transcription 20-40 fold in lens cells, it covers nearly 1kb with a 120bp core region, and it will operate independently of position and orientation.

There are two types of α-crystallins, αA and αB, which are very similar. The chicken αA gene has been cloned and consists of three exons occupying 4.5kb of DNA (Thompson et al., 1987). The 5' flanking region contains a consensus sequence $\mathrm{GCAGCA^{T}{}_{G}CCCTCC^{T}{}_{A}G}$ present in most crystallin genes (Thompson et al., 1987). A sequence -242 to -189 from the cap site was found to be responsible for high expression in lens cells when a hybrid gene was transfected into them (Okazaki et al., 1985).

The ß-crystallins are a complex family of polypeptides comprising about 50 percent of lens proteins. There are at least six major species and perhaps as many as 18 altogether. cDNAs for ß-crystallin have been prepared from chicken lens. Four different clones selected the mRNAs for the six major polypeptides; each clone represented a different gene (Hejtmancik and Piatigorsky, 1983). The gene for one of these polypeptides, ßB1 (ß35), is interesting because it is expressed specifically in elongating lens cells and not in the central cuboid epithelial cells; its DNA sequence has been determined (Hejtmancik et al., 1986).

The sequencing of the lens crystallins identified some surprising similarities to a spectrum of metabolic enzymes. For example, δ-crystallin and the enzyme arginosuccinate lyase of the urea cycle are strikingly similar, and duck δ-crystallin has a very high ASL activity although chicken is much lower (Piatigorsky et al., 1988). The human ASL gene hybridizes with the chicken δ1 crystallin gene and especially the δ2 gene; these are the only genes in the chicken that hybridize with the human ASL gene, now suggesting a role for the δ2 gene in coding for ASL (Piatigorsky et al., 1988).

CYTOSKELETON GENES: TUBULIN AND VIMENTIN

Tubulin. Microtubules are filamentous polymers used in a variety of cellular processes including maintaining intracellular cytoplasmic structure and transport; they are composed mainly of heterodimers of one α and one ß-tubulin subunit. Using cDNA probes, there were reported to be 4-5 genes coding for α-tubulin and 4-5 for ß-tubulin in chickens (Cleveland et al., 1980, 1981). Four gene segments were cloned for ß-tubulin and they initially appeared to be the complete set in the chicken genome, each coding a single distinct mRNA (Lopata et al., 1984). These gene-specific probes were used to analyze the expression of the four mRNAs between tissues and in development (Havercroft and Cleveland, 1984). For example, the ß1 gene transcript is a minor component in all cells and tissues; ß2 transcripts are present in large amounts in embryonic brain and spinal cord neurons; ß3 is found at high levels in the testis although in lower amounts in other tissues;

ß4' (later ß7) is present at moderate levels in all tissue types. All seven chicken ß-tubulin genes have now been cloned and sequenced and their expression studied (Sullivan and Cleveland, 1984, 1986; Sullivan et al., 1985, 1986a, 1986b; Murphy et al., 1987; Monteiro and Cleveland, 1988). The ß-tubulins are a family of distinct evolutionarily conserved isotypes with a characteristic carboxyl-terminal variable region, and to a lesser extent, an amino-terminal variable region; all the genes are divided into four exons (Sullivan and Cleveland, 1986; Sullivan et al., 1986b). The protein coding regions of the ß1, ß2, ß3, and ß4 genes are highly conserved; the derived amino acid sequence of ß1 differs from ß2 at 0.5 percent (Sullivan et al., 1985), from ß3 at four percent (Sullivan et al., 1986b), and from ß4 at 8.9 percent (Sullivan and Cleveland, 1984). There is, however, no homology in the 5' or 3' flanking sequences (Sullivan et al., 1986b). The ß5 gene encodes a more divergent polypeptide distinguished by two discrete variable regions, although it has sequences in common with the other divergent chicken gene, ß4, suggesting two families of tubulin genes (Sullivan et al., 1986a). The expression of ß4 and ß5 is complementary; ß5 is expressed in a wide variety of cell types except neurons, and ß4 is uniquely expressed in neurons (Sullivan et al., 1986a). The maximum sequence divergence among those ß-tubulins is eight percent. The tubulin of erythrocytes, ß6, has been cloned and sequenced, it is 17 percent divergent in its amino acid sequence, and it is the major isotype of erythrocytes. The seventh tubulin, ß7, has also been recently cloned and is highly expressed in all cell types (Monteiro and Cleveland, 1988). The seven genes are expressed as follows: ß1 is an abundant muscle subunit, ß2 is a major brain subunit, ß3 is a widely expressed subunit that is the dominant species in testis, ß4 is expressed exclusively in neurons, ß5 is expressed at low levels in all cells except neurons, ß6 is the major subunit in erythrocytes, and ß7 is expressed at moderate levels in most cells (Monteiro and Cleveland, 1988).

Vimentin. Vimentin is a member of the intermediate filament group of proteins. cDNA for vimentin was isolated, and although there are two abundant mRNA species (2.0 and 2.3kb), the gene was a single copy (Zehner and Paterson, 1983a). Both transcripts are functional *in vivo*, differing in their 3' untranslated sequences; they are both transcribed at similar rates in most tissues (Zehner and Paterson, 1983b); embryonic erythroid cells express predominantly the lower molecular weight RNA (Capetanaki et al., 1983). The vimentin gene has been sequenced and is conserved compared with the hamster gene (74 percent nucleotide, 80 percent amino acid). There are regions where the homology is absolute, and there are conserved sequences in the 5' flanking region which are suggestive of regulatory sequences (Zehner et al., 1987).

GLYCOLYSIS AND ASSOCIATED PATHWAYS

A number of genes encoding these enzymes have been cloned in chickens, but little or no detailed analyses have been performed. Where studies of structure and expression have been made, they are considerably less advanced than for the equivalent genes in mammals.

Pyruvate kinase. The gene for chicken PK has been cloned and analyzed. It has at least ten introns which divide the coding sequence into discrete elements of secondary structure, and their position seems to be evolutionarily conserved (Lonberg and Gilbert, 1985).

Triosephosphate isomerase. The cDNA encoding for TPI was isolated from a muscle cDNA library with radiolabelled cDNA from RNA that had been enriched by polysome immunoselection. The natural gene was isolated from a genomic DNA library. It complements an *E. coli* mutant deficient in TPI when placed downstream from a hybrid *trp-lac* promoter (Strauss and Gilbert, 1985a). The genomic clone has been sequenced, and is 3kb long with seven exons which appear evolutionarily conserved (Strauss and Gilbert, 1985b).

Malic enzyme. The cDNA encoding malic enzyme has been isolated from a goose uropygial gland cDNA library. This cDNA cross-hybridized with chicken and showed that nutritional and hormonal stimulation of hepatic malic enzyme synthesis in both chickens and geese was regulated pretranslationally (Winberry et al., 1983).

Phosphoenolpyruvate carboxykinase. A cDNA clone was isolated from a kidney cDNA library (Hod et al., 1984a). The chromosomal gene isolated from a genomic library is 8kb long, and it has eight exons with two mRNAs having different 5' ends whose ratios are the same in different tissues and after hormone induction (Hod et al., 1984b).

Aldolase B. This gene has been isolated and sequenced from a genomic library. It is 8.7kb long and has eight introns. There were no differences in RNA structure between tissues (Burgess and Penhoet, 1985).

Fatty acid synthetase. A cDNA encoding this enzyme was isolated from a goose uropygial gland cDNA library. The mature FAS mRNA is large, being about 16kb. The nutritional regulation of FAS synthesis occurs at a pretranslational level (Morris et al., 1982).

OTHER ENZYMES

Cytochrome C. Compared with 20-30 sequences in mammals, only a single gene has been isolated from a genomic library, although there are two alleles differing in their 5' flanking sequences (Limbach and Wu, 1983).

Cytochrome P-450. A cDNA for this protein has been isolated. It is 45-50 percent conserved compared with mammals, and is a member of a group of four distantly related gene subfamilies (Hobbs et al., 1986).

Carbonic anhydrase II. There are three loci encoding the isoenzymes CAI, CAII, and CAIII. cDNA for CAII has been isolated from an anemic chicken erythrocyte cDNA library (Yoshihara et al., 1985).

Metallothionein. A cDNA library was constructed from liver RNA of chickens treated with zinc, and screened with a mouse MT-I probe. A 376bp cDNA clone was isolated which was 70 percent homologous to a consensus sequence of mammalian DNA (Wei and Andrews, 1988). Using this cDNA it was shown that the chicken MT RNA level responds to a variety of stimuli within and between tissues. There seems to be a single isoform of MT in chickens.

Glutamine synthetase. A cDNA encoding this enzyme was isolated from a chicken retina cDNA library. The developmental profile of this enzyme was reflected in mRNA levels (Patejunas and Young, 1987).

Thymidine kinase. The chicken TK gene was isolated by plasmid rescue on the basis of its ability to produce TK in mouse L tk^- cells (Kwoh et al., 1983). The gene has been sequenced; it is divided into seven small (about 160bp) exons by six small (230 bp) introns (Kwoh and Engler, 1984).

5-aminolevulinate synthase. Both cDNA and genomic clones for ALV synthase have been isolated. In chickens it is encoded in a single gene (Elferink et al., 1987). The gene has been sequenced and is 6.9kb long with ten exons (Maguire et al., 1986). A conflicting report has, however, suggested that there are several tissue and developmentally-regulated mRNAs, although they are only similar, encoded in a family of closely related genes (Yamamoto et al., 1985).

OTHER GENES

Only some of the more interesting chicken genes and gene families are discussed here. A more comprehensive list is included in Table 23.1. An updated version can be obtained from the EMBL and GenBank databases.

Heat shock proteins. The heat shock proteins are highly conserved. Antibodies raised to the chicken 89K, 70K, and 24K proteins crossreact with antigenic material in widely different species from yeast to mammals (Kelley and Schlesinger, 1982). A clone for the HSP70 gene was isolated from a genomic library by crosshybridizing to the *Drosophila* HSP70 gene; it contains an open reading frame of 1.9kb which predicts a 70kD protein, and it has upstream TATA and CCAAT boxes, a SP1 binding site, and two overlapping heat shock regulatory elements (Morimoto et al., 1986).

A novel heat shock protein, HSP108, which is inducible by steroids, has been isolated from chicken oviduct (Sargan et al., 1986). cDNA and genomic clones have been obtained. The gene is 9.9kb long and is split into 18 exons (introns are rare in HSP genes). It has upstream TATA and CCAAT boxes and a putative steroid hormone binding site (Sargan et al., 1986; Kleinsek et al., 1986).

Ubiquitin. This is an evolutionarily high conserved protein, which conjugates with other proteins, 'tagging' them for proteolytic degradation. Using heterologous probes, two different chromosomal chicken genes were isolated, UbI and UbII (Bond and Schlesinger, 1986). UbI contains four copies of the protein coding sequence in tandem, and UbII contains three. Two mRNAs are found after heat shock of chicken embryo fibroblast cells, both from the UbI gene. The mRNA is unstable suggesting a specialized and transient role for ubiquitin during heat shock.

Cell adhesion molecules. cDNA for both neural-CAM and liver-CAM have been isolated (Gallin et al., 1985; Murray et al., 1986). Both cDNA and genomic clones for calcium dependent L-CAM have been isolated and used to demonstrate that the same mRNA is synthesized in eight embryonic tissues (Gallin et al., 1985). Sequences analysis of L-CAM cDNA suggests that it is the result of internal gene duplication and not similar in sequence to N-CAM (Gallin et al., 1987). The cDNA isolated for N-CAM has shown that there are two mRNA species, and hence the two proteins of different size are derived by splicing of transcripts from a single N-CAM gene (Murray et al., 1986). This calcium dependent N-CAM has been sequenced and shown to have an extracellular transmembrane and cytoplasmic domain, and to be a member of the cadherin gene family which comprises epithehal, placental, and now neural-cadherin (Hatta et al., 1988).

Fibronectin. Cellular fibronectin is a glycoprotein present on the surface of many cells. Both cDNA and genomic sequences for the chicken protein have been cloned. It is a large gene of 48kb with at least 48 exons (Hirano et al., 1983). A 4.3kb region has been sequenced; the cell binding domain consists of many repeats of small units of amino acids (Kubomura et al., 1987).

Miscellaneous genes. The chicken gene for *preproinsulin* has been cloned. It is present in a single copy in the genome and has two introns (Perler et al., 1980).

The *embryonic pepsinogen* gene is 3.5kb long with nine exons. It is believed to be the homologue of the mammalian prochymosin gene (Hayashi et al., 1988).

The *ß-nerve growth factor* gene shows 85 percent amino acid homology with mammals although the pattern of expression differs somewhat (Ebendal et al., 1986). The 3' end of the *epidermal growth factor receptor* gene overlaps the c-*erbB* locus and is homologous with the human gene; the gene generates six different mRNA transcripts (Olofsson et al., 1986).

The cDNA for a unique membrane glycoprotein, LEP100, which shuttles from lysosomes via endosomes to the plasma membrane and back, has been cloned (Farnbrough et al., 1988).

The gene for the H-subunit of the ubiquitous iron storage protein *ferritin* has been cloned. It only exists in a single copy in the chicken genome. It is 4.6kb long with four exons. It has 93 percent amino acid identity to the human gene (Stevens et al., 1987).

There is a cluster of genes coding for *feather keratin.* Five genes have been isolated as part of a cluster (Molloy et al., 1982).

THE FUTURE

The molecular genetics of chickens has so far concentrated on genes that were more easily analyzed from chicken material, for example the steroid inducible genes of egg white and yolk and the muscle protein genes, or on genes whose main interest lies in evolutionary comparison with mammals, for example the globins and the collagens. In the future it is likely that chicken genes will be cloned and analyzed more for their own sake in the context of the chicken as a commercially important animal for human food.

With the imminent development of routine production of transgenic chickens (see Chapter 24) it will become important to identify the genes controlling the major production traits in chickens such as muscle growth, fertility (egg production), and disease resistance. The identity, number, and nature of these 'trait genes' and their products is almost totally unknown. Furthermore the products of only about one percent of the estimated 100,000 expressed genes in higher organisms are known; these are mainly the enzymes of intermediary metabolism and the protein hormones. Therefore to make full use of transgenic technology, this huge gap in knowledge has to be filled and strategies have to be devised to identify trait genes and their products.

There is no easy short-cut to solving this problem. Gene identification can be approached from the trait downwards, attempting to dissect physiological processes toward individual gene products by making use of strains of animals genetically divergent in the target trait (Bulfield, 1985). Or, gene identification can be approached from the gene upwards, making use of restriction fragment length polymorphisms (RFLPs) or two dimensional gel electrophoresis of gene products (Bulfield, 1985), again making use of genetically divergent animals.

Unfortunately in chickens both these approaches have had little attention and a lot of basic work is required. For example, there are few known RFLPs in the chicken. It is this area of gene identification for commercial traits that must be the challenge of the next few decades for chicken molecular geneticists.

Table 23.1. Cloned chicken genes.

acetylcholine nicotine receptor (δ subunit)
acetylcholine nicotine receptor (γ subunit)
α-actin (cardiac muscle)
α-actin (skeletal muscle)
α-actin (smooth muscle)
ß-actin (cytoplasmic)
ß-actin (embryonic cytoplasmic)
actin type 5 (cytoplasmic)
adenylate kinase (muscle)
albumin (serum)
aldolase B
apolipoprotein II (apoVLDLII)
aspartate aminotransferase (mitochondrial isoenzyme)
BS1a protein binding site (5' to lysozyme gene)
BS1b protein binding site (5' to lysozyme gene)
B2S protein binding site (5' to lysozyme gene)
calcitonin
calcium-binding protein
calmodulin
cartilage matrix protein
chondrocyte specific short collagen (encoding HMW/LMW precursor)
creatine kinase-B
creatine kinase-M
cCM1 (calmodulin-like protein)
CR1CMa,b,c,d; CR1 repetitive element (downstream of cCM1 gene)
histone H1
histone H2A
histone variant H2A.f
histone H2B
histone H3
histone H4
histone H5
histone LH4, RH4, H3, LH2A and RH2A
histone erythrocyte-specific H5
α-globin
ß-globin
c-mht (analogue to v-mht oncogene)
c-mil (proto-oncogene)
c-myc (proto-oncogene)
c-myb gene (analogue to v-myb oncogene)
collagen α-1 type -1
collagen α-2 type -1
collagen type-1 gene
collagen α-1 type II
collagen α-1 type III
collagen α-1 type IX
collagen α-2 type IX
conalbumin
CPS1 processed (pseudogene related to ras oncogene)
criova (middle-repetitive sequence)
criuia (middle-repetitive sequence)
c-ros (proto-ongogene)
crystallin α
crystallin δ-1
crystallin δ-2
c-src (proto-oncogene)
cytochrome c
cytochrome P-450 (phenobarbital-inducible)
desmin (intermediate filament protein)
erbB (homologue of AEV v-erbB oncogene)
ev-1 (retrovirus integration site)
fibronectin
fps (proto-oncogene)
ß-galactoside-binding lectin
glyceraldehyde-3-phosphate dehydrogenase
ovomucoid
phosphoenolpyruvate carboxykinase (GTP)
protease (calcium)
pyruvate kinase (muscle)
reverse transcriptase-related DNA (ev-segment 1 and 2)
7S RNA
spectrin-nonerythroid α
suppressor tRNA
thymidine kinase cytoplasmic
transforming gene

Continued ...

Table 23.1. Cloned chicken genes. (Continued)

embryonic ρ (ß-like) globin
embryonic ε (ß-like) globin
heat shock protein (70 kD)
heat shock protein (108 kD)
Ig germline λ-chain J region
Ig germline λ-chain VI
Ig germline λ-chain psi-VI pseudogene
Ig germline λ-chain psi-V2 pseudogene
Ig germline λ-chain psi-V3 pseudogene
Ig λ light chain
Ig heavy chain (secreted μ constant region)
insulin (peproinsulin)
integration site (for spleen necrosis virus)
keratin feather B
keratin feather C
keratin scale III
lysozyme (egg white)
lys-tRNA (embryo)
mitochondrial matrix protein
  (5-aminoiaevulinate synthase) mRNA
myosin alkali 1-chain a1
myosin alkali 1-chain a2
mysoin heavy chain
myosin embryonic heavy chain
myosin fast alkali light-chain
myosin light chain 2A
myosin light chain 2 (LC2f)
neural cell-adhesion molecule (N-CAM)
ovalbumin
  (homologous to transferrin family)
triosephosphate isomerase
tropomyosin smooth-muscle α
tropomyosin major α
tropomyosin minor α
troponin T
tubulin ß-4
tubulin ß-5
tubulin brain
U1 small nuclear RNA
U1 small nuclear RNA (pseudogene)
U2 small nuclear RNA
U4A small nuclear RNA
U4B small nuclear RNA
U4C small nuclear RNA
U5A small nuclear RNA
U5B small nuclear RNA
ubiquitin
  (from heat-shocked embryo cells)
vimentin
  (intermediate filament protein)
vitellogenin II
X gene
Y gene

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Chapter 24. GENETIC ENGINEERING

Ruth M. Shuman

Genetic engineering is a new tool for genetically improving poultry. It emerged in the early 1980s as a result of discoveries in molecular genetics and is based on the understanding of DNA: how genetic information is stored, how it is used to determine the structure of proteins, and how it is transmitted from generation to generation. Through this understanding, technology has developed to permit direct examination of DNA. This includes the ability to transfer genetic material from one organism to another and to use cloned genes to develop a new class of genetic markers. Only recently has this technology begun to be applied to poultry breeding.

Current poultry breeding practices depend on natural genetic variation and sexual reproduction for genetic improvement. Poultry breeders use natural variation as the starting material for selection, and each parent contributes half its genetic material to the next generation. Selection is based on genetic merit and is used to improve genetic characteristics within a breeding population. New genetic material is incorporated from mutations or by crossing and backcrossing to populations containing desired traits.

The idea is to use gene transfer and genetic engineering as aids to enhance current breeding practices. By permitting artificial incorporation of foreign genetic material directly into the genome, gene transfer will contribute to breeding methods by improving genetic characteristics of poultry. Once foreign genes are incorporated, breeders will need to follow a selection and propagation strategy to optimize the effect of these genes in their breeding program. Therefore, gene transfer will not replace current breeding practices, but they will add to present technology.

Genetic engineering offers potential benefits, such as providing a means to increase genetic variation, and producing genetic change more rapidly in poultry populations. By directly inserting genes, breeders will bypass reproductive barriers and permit gene flow between vastly different organisms as well as between different breeding populations of the same species. The result will be new genotypes with possible increased economic value. Gene transfer may also reduce the current effort required to transfer favorable traits from low-performing to high-performing stocks. Moreover, it may be possible to use DNA analysis to identify desirable genotypes in birds to be selected as breeders for the next generation, thus reducing reliance on progeny testing.

The goal of this chapter is to describe briefly current gene transfer technology and related molecular methodology, to summarize progress in the area of genetic engineering, to present methods for evaluating and introducing transgenic birds into breeding populations, and to discuss future applications and implications related to genetic engineering in poultry. Even though written from a practical poultry breeding standpoint, this chapter includes perspectives for using this technology for basic research and other future applications such as developing new biological production systems.

GENE TRANSFER OVERVIEW

Introducing foreign DNA is now routine in the mouse (see review by Palmiter and Brinster, 1986) and it has been accomplished successfully in domestic mammals including pigs, sheep, and rabbits (Hammer et al., 1985). It is also possible to transfer genes to avian genomes (Souza et al., 1984; Shuman and Shoffner, 1986; Salter et al., 1986). However, genetic engineering technology in poultry is still in the early stages of development (reviewed by Bulfield, 1985; Freeman and Messer, 1985; Shoffner, 1986; Crittenden and Salter, 1986; Freeman and Bumstead, 1987). Research to date has primarily focused on finding the best method to transfer foreign genes. Several approaches are described in the following pages.

In addition, there is much ongoing basic research in molecular biology of avian and other animal genes, although its main emphasis is not necessarily using these genes for genetically improving poultry. It is estimated that there are 50,000-100,000 genes in the avian genome. Thus far, only about 100 have been cloned which represents but a small fraction of the potentially useful genes to be identified in the future (see Table 23.1).

Successfully using genetic engineering technology in poultry will depend on several factors. First, it will be necessary to develop a methodology to produce transgenic birds, those with foreign genes transmitted through the germ line, routinely and in high numbers. This is essential as work in other animals indicates that there is much phenotypic variability associated with the transferred gene. Transgenic birds possessing the desired characteristics may occur infrequently. Second, it will be necessary to identify, isolate, and clone useful genes. However, perhaps more importantly, it will be crucial to understand the function of those genes to be applied to poultry improvement before they can be utilized successfully. Transferred genes may also require modifying to obtain the desired effect. Further, regulating expression of the transferred gene will be another important consideration. Thirdly, birds containing transferred genes and identified as expressing the desired trait will need to be tested thoroughly to determine the stability of the new genotype and phenotype. Also, it will be necessary to determine if other phenotypic changes have occurred due to the transferred gene and if these changes cause an overall decrease in phenotypic value. Finally, testing must ensure that transgenic birds and the methods used to produce them pose no risk to poultry production, consumers, or the environment. Having met these requirements, genetically engineered poultry may then be used in breeding programs.

GENE TRANSFER METHODOLOGY

Target cells. Several cell types have been proposed as potential targets for inserting DNA. The primary consideration in choosing a target cell has been one which will provide the most efficient and direct transfer of new genes into the genome of germ line (reproductive) cells. Accessing the germ line may occur through either reproductive cells such as spermatozoa (Pandey and Patchell, 1982), ova (Shuman, 1984), and primordial germ cells (Shuman, 1981), intact early embryos (Salter et al., 1986), isolated early embryonic cells or embryonic stem cells (pluripotent cells isolated from the inner cell mass of the blastocyst) (Bradley et al., 1984).

Directly inserting DNA into reproductive cells or embryos at the one-celled stage of development means that the transferred gene will incorporate into all of the somatic and germ line cells of the resulting bird. This reduces the frequency of germ line mosaicism, an advantage in that transgenic birds may be identified in the parent (G-O) generation. However, the efficacy of producing transgenic birds may limit using this approach.

Insertion strategies using embryos that have more than one cell or which involve producing chimeras with embryonic cells or embryonic stem cells will result in mosaics.

These mosaics will contain either cells that have not incorporated the transferred gene, or cells that contain the transferred gene at different loci or in varying copy number. A disadvantage in producing mosaics is that identifying transgenics requires mating and examination of the next generation (G-1). This is difficult if the proportion of germ cells containing the foreign gene is low. There is, however, a genetic advantage in obtaining germ line mosaics. When mated, a mosaic may produce progeny that each has a different genetic potential based on the insertion site of the new gene. This may, especially for male mosaics, result in increasing the number of transgenic birds that can be produced for testing and evaluation. Also, it may increase as well the probability of identifying birds with the desired phenotypic characteristic.

Another approach being considered is targeting somatic cells for gene insertion. Conceivably, gene transfer may be used to produce a transient (one generation) effect which is not intended for germ line transmission by altering the genotype of somatic cells. In the future, for example, it may be possible to introduce genes at the final commercial cross that tailor production to specific needs without developing and maintaining additional lines of birds. In this case, the target for gene insertion may be the embryo at a multiple cell stage of development or a specific tissue based on the application.

DNA delivery methods. Potentially several methods exist for transferring new DNA into cells; these include enhancing the uptake of DNA with chemicals (Graham and van der Eb, 1973; Kawai and Nishizawa, 1984) or liposomes (Schaefer-Ridder et al., 1981), microinjecting DNA (Graessman and Graessman, 1976), and using DNA (Goff and Berg, 1976; Mulligan and Berg, 1980) and RNA (Shimotohno and Temin, 1981; Wei et al., 1981; Tabin et al., 1982) viral vectors. Transferring DNA to cultured avian cells presents little difficulty. However, with the exception of retroviral vectors (derived from RNA viruses), these methods have not been successful for transferring genes to avian embryos. This failure may result from the difficulty in handling and microinjecting the large fragile avian embryo (Shuman, 1984; Shuman and Shoffner, 1986), coupled with the low frequency of stable integration of microinjected DNA (Brinster et al., 1985). Also, it could result from the arduous task of obtaining significant numbers of hatched chicks following embryo manipulation (Perry, 1988).

The most effective methods for transferring DNA to the germ line of the mouse and other mammals have been either by direct microinjection of DNA into the pronucleus of the one-celled embryo (Gordon et al., 1980; Hammer et al., 1985) or by infecting embryos with retrovirus vectors (Soriano and Jaenisch, 1986). In contrast, DNA viral vectors (derived from DNA viruses) have been useful in developing new delivery systems for vaccines and in other cases where primarily transient expression is required (Baichwal and Sugden, 1986). These viruses have not been used widely, however, to introduce DNA into the germ line because they do not possess an efficient method to integrate into the host's chromosomes.

Retrovirus vectors overcome the difficulties encountered when transferring genes to avian embryos and have become the method of choice for avian gene transfer. They have been selected as vectors because of their unique infection cycle. Upon infecting the host, these RNA viruses produce a DNA intermediate that integrates into the host's genome (Temin, 1986). Thus, retroviral vector-based gene transfer technology takes advantage of the virus infection cycle.

Briefly, the infection cycle occurs in two phases (Varmus and Swanstrom, 1984, 1985; Varmus, 1988). In the first phase, encompassing DNA synthesis and integration, the retrovirus is adsorbed to the cell aided by cell surface receptors that recognize the viral envelope glycoprotein. Upon entering the host cell cytoplasm, the viral RNA genome is used to synthesize a DNA copy through the activity of the viral enzyme reverse transcriptase. Then, the viral DNA integrates into the host's DNA at viral sites located at the termini of the long terminal repeat (LTR) region. Called a provirus, this integrated viral DNA is found to insert randomly along the host's chromosomes. The second phase of the infection cycle involves the replication of new virus particles (Dickson et al., 1984, 1985). The provirus uses the transcriptional and translational apparatus of the host cell to produce viral RNA and proteins that combine to create infectious virus particles. New virus particles are then free to begin a new round of infection.

Therefore, using retroviruses to deliver DNA provides the advantage of an efficient and nontraumatic method to enter the host cell and to integrate into the host genome. In addition, proviruses typically number one per integration site and one or two per cell (the number per cell may depend on the titre of the virus stock used for the infection and other viral and host factors). Thus, the number of copies of transferred genes per cell is similar to that found normally for unique host genes. Also, the viral DNA integrates into the host DNA at a specific site in the viral genome. This reduces the chance of recombination in the transferred gene, a situation which often occurs when DNA is inserted by other methods such as microinjection.

There are two types of retrovirus vectors, those constructed from nondefective viruses which can replicate in the host cell, and those constructed from defective viruses designed to complete only the first phase of the infection cycle and are unable to replicate. Nondefective retrovirus vectors contain, in addition to the transferred gene, all the necessary viral sequences to produce a virus particle. These include the *trans*-acting regions that encode the viral proteins and the *cis*-acting regions that encode other sequences necessary to replicate. Three main *trans*-acting regions produce the viral proteins. First, the *gag* region encodes the structural and processing proteins of the core of the virus; next, the *pol* region encodes the viral DNA synthesis and integration enzyme activities, and finally, the *env* region encodes the envelope glycoproteins. Especially important *cis*-acting sequences are the encapsidation or packaging site that allows the viral RNA to be packaged into the virus particle, the genetic regulatory sequences (promoter and enhancer) that control expression of the virus, the termination signal which locates the 3' end of the virus, the primer binding site which initiates viral DNA synthesis, and the sequences that are required for integration of the viral DNA into the host's chromosomes (Varmus and Swanstrom, 1984, 1985).

Defective retrovirus vectors are missing either *trans*- or *cis*- and *trans*-acting regions of the virus and, therefore, cannot produce virus particles (Figure 24.1). Vectors must retain the encapsidation site and other *cis*-acting regions, however, to ensure efficient packaging and integrating of the vector. The simplest of the defective vectors replaces the viral protein coding region with the gene to be transferred. Recently, retrovirus vectors have been constructed that contain additional deletions in the 3' viral promoter and enhancer region (Yu et al., 1986). These vectors, referred to as self-inactivating or suicide vectors, lose the viral regulatory sequences in the vector DNA that integrates into the host genome. This loss precludes transcription of vector RNA in host cells, leaving only the transferred gene and its regulatory sequence active.

Because the defective vectors cannot replicate, vector virus is produced in specially constructed cells, called helper cells (Watanabe and Temin, 1983). Helper cells are designed to produce pure vector virus stock free from contamination of nondefective (helper) virus (Figure 24.1). These cells complement the defect in defective vectors by supplying the *trans*-acting proteins of the *gag*, *pol*, and *env* regions of the virus. Helper cells contain the viral protein coding region. However, to reduce the possibility of contamination with

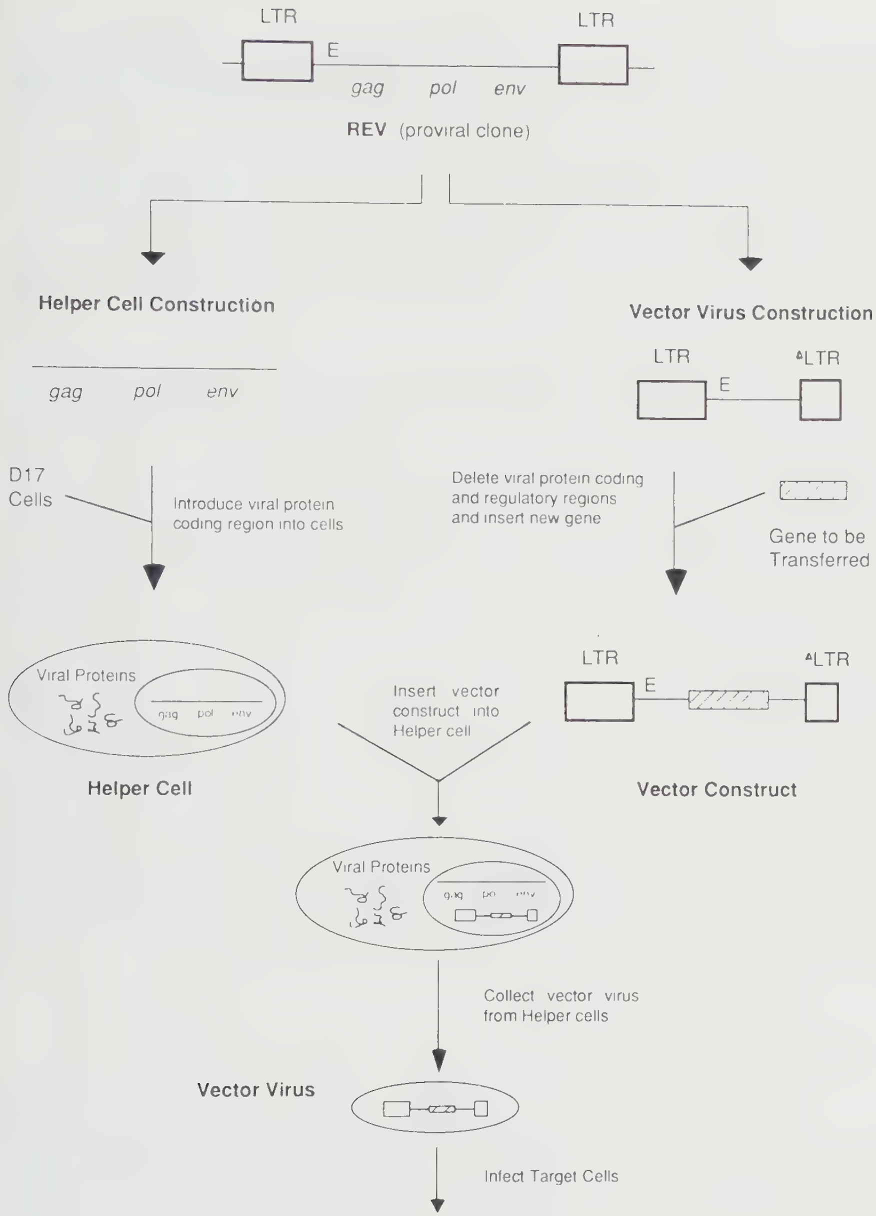
LTR
LTR
E
gag
pol
env
REV (proviral clone)
Helper Cell Construction
Vector Virus Construction
gag
pol
env
LTR
ΔLTR
E
D17 Cells
Introduce viral protein coding region into cells
Delete viral protein coding and regulatory regions and insert new gene
Gene to be Transferred
Viral Proteins
gag
pol
env
LTR
ΔLTR
E
Insert vector construct into Helper cell
Helper Cell
Vector Construct
Viral Proteins
gag
pol
env
Collect vector virus from Helper cells
Vector Virus
Infect Target Cells

Figure 24.1. Diagram of the production of defective vector virus in helper cells. The starting material is a proviral clone of the reticuloendotheliosis virus (REV). Helper cells are produced by inserting the viral protein coding region (*gag*, *pol* and *env*) into the canine transformed cell line D17. Vector is produced by deleting the viral protein coding region and the 3' promoter and enhancer (LTR = long terminal repeat and contains viral regulatory element, ΔLTR = deleted promoter and enhancer) and inserting the gene to be transferred. The encapsidation sequence (E) is retained in the vector construct. Vector virus containing the gene to be transferred is produced following insertion of the vector construct into the helper cells and may be used to infect target cells.

helper virus, they are missing the encapsidation site to preclude the packaging of helper virus into virus particles. Further, in improved helper cell lines that also lack other *cis*-acting regions, inadvertent generation of nondefective virus can be eliminated (Shuman, unpublished).

Nondefective vectors are useful for transferring genes to cultured cells or embryos where the presence of replicating virus does not present a hazard or interfere with the expression of the transferred gene. Because they can replicate and spread from cell to cell, these vectors have the advantage of a relatively high rate of infection not dependent on titre of the vector virus stock produced. Their main disadvantage is that they may be pathogenic or may lead to forming new pathogenic virus strains in transgenic birds as well as decreased immune response to retrovirus infection.

Defective vectors, on the other hand, due to their inability to replicate, may be preferred for transferring genes to the germ line or for commercial somatic cell applications. If improved helper cells found to be free of helper virus are used to produce defective vectors, the chance of producing pathogenic virus is removed. Using self-inactivating vector virus, moreover, reduces the possibility of expression of the vector sequences in the host cell. This further improves safety by decreasing the probability that the vector will recombine with related viruses encountered in the field. Deleting the viral regulatory sequences in self-inactivating vectors also helps to ensure that the vector sequences do not alter the expression of the transferred gene. Primarily, the disadvantage of defective vectors is that the titre of the virus stock produced is low, thus decreasing the efficiency of transferring the inserted gene.

Although retrovirus vectors are the preferred method for avian gene transfer, several limitations accompany this system. Foremost is the extra effort required to produce viable high-titre vector virus stock. Each vector construct must be tested to determine that the inserted gene does not interfere with producing vector virus. In addition, retroviral vectors theoretically can transfer up to ten kb of genetic material (Temin, 1986). However, as the size of the vector increases, the titre of the vector virus stock produced proportionally decreases. Another limitation is the inability to control the site of integration of the vector sequence in the host chromosome, although gene targeting has been shown to be possible in embryonic stem cells by Thomas and Capecchi (1987). Undoubtedly, researchers will continue to search for improved methods to transfer genes in poultry.

REVIEW OF POULTRY GENE TRANSFER RESEARCH

Retrovirus vectors were first used to transfer genes to cultured cells. Defective vectors have been constructed from the reticuloendotheliosis virus (REV) group (for review see Temin, 1986). Early vectors were used to transfer successfully the herpes simplex thymidine kinase gene or other marker genes and required helper virus for vector virus production (Shimotohno and Temin, 1981). Shortly thereafter, a REV helper cell line was produced that facilitated producing vector virus stock (Watanabe and Temin, 1983). Nondefective vectors have been constructed from Rous sarcoma virus (Hughes and Kosik, 1984). In these vectors, the region encoding the *src* oncogene has been removed and replaced with a synthetic DNA linker to allow insertion of a foreign gene. These vectors have been used to transfer the chloramphenicol acetyltranferase (*cat*) marker gene to cultured chicken cells (Greenhouse et al., 1988).

A REV vector containing the neomycin resistance gene and the herpes simplex thymidine kinase gene has been used to examine the production of transgenic poultry by ova infection (Shuman, 1984; Shuman and Shoffner, 1986). Helper cells producing vector virus were injected into developing ovarian follicles near the time of ovulation in the chicken. Hatched chicks that contained vector sequences in their genomic DNA were identified by Southern blot hybridization. Ovarian infection has also been shown to occur with nondefective REV (Shuman 1984; Shuman and Shoffner, 1986). Twenty-six percent of the progeny that developed following intrafollicular inoculation with REV showed evidence of virus infection while one percent exhibited infection following inoculation outside the follicle.

More recently, a defective REV vector was used to transfer the *cat* gene to early Japanese quail and chicken embryos (Shuman et al., 1988). Vector virus, or virus-producing cells, were injected into embryos prior to incubation. In these studies the *cat* gene, expressed from the viral regulatory elements, was detected in a wide variety of tissues. These included the bursa of Fabricius, brain, heart, intestine, liver, skeletal muscle, spleen, and thymus. In addition, CAT enzyme activity was observed in early, middle, and late embryonic development and in posthatch chicks.

Souza et al. (1984) were the first to report the transfer of an exogenous gene to chicken embryos using a nondefective vector derived from Rous sarcoma virus. They transferred the chicken growth hormone gene by inoculating embryos incubated to nine days. Growth hormone levels were found to be elevated in the serum of some birds following hatching. Growth rate, however, was not significantly different from control birds.

Bosselman et al. (1989) have shown that defective retrovirus vectors also may be used to transfer foreign genes to the germ line cells of the chicken embryo. Unincubated embryos were inoculated with a REV vector virus containing the herpes simplex thymidine kinase gene and the neomycin phosphotransferase gene. Eight percent of the males that hatched contained vector DNA in their semen and transmitted vector sequences to their progeny.

In addition, it has been shown that nondefective recombinant avian leukosis viruses (ALV) and REV can be transmitted through the germ line following infection (Salter et al., 1986, 1987; Crittenden et al., 1988). Unincubated chicken embryos or hatched chicks were inoculated with virus, and then the resulting progeny were tested by DNA analysis to determine whether they produced the virus and whether they could genetically transmit proviral DNA to their progeny. Viremic parent males were found to transmit integrated proviral DNA to their progeny at frequencies of 1-11 percent. Subsequently, the progeny were observed to transmit the proviral DNA in a Mendelian fashion to the next generation, a positive indication of germ line transmission. Interestingly, Japanese quail have been shown to be resistant to infection and germ line transmission of nondefective ALV (Salter, Hughes, and Bradac, 1988, personal communication; Shuman, unpublished).

Further, Salter and Crittenden (1988) also have described a chicken line produced in the above study that contains a defective ALV. This line has been shown to express only the envelope glycoproteins (type subgroup A) of the virus. Chicken embryo fibroblasts prepared from this chicken line display a high degree of viral interference to subgroup A Rous sarcoma virus infection. In addition, the chickens from this line have proved to be resistant to ALV infection. This resistance demonstrates that the envelope gene acts as a dominant gene for resistance to ALV infection and may be transferred to the germ line by retroviral vector.

An alternative method for introducing genetic material, using irradiated sperm, has been reported (Pandey and Patchell, 1982; Bumstead et al., 1987; Tomita et al., 1988). In these studies, hens were first inseminated with irradiated semen obtained from a donor male carrying marker traits. Then, the hens were reinseminated 24 h later with nonirradiated semen obtained from males of the same strain as the hen. Using this method, Pandey and Patchell (1982) and Tomita et al. (1988) showed that 3-5 percent of the resulting progeny had feather and egg color characteristics of the marker male. This result was confirmed by Bumstead et al. (1987), who found that 0.4 percent of the resulting progeny carried the major histocompatibility haplotype marker of the irradiated sperm in addition to the haplotypes of the dam and nonirradiated sire. In contrast, Shoffner et al. (1987) and Shoffner (1988, personal communication) have reported that irradiation of semen does not result in the transfer of marker traits.

Spermatozoa have also been examined as a possible DNA delivery system for the production of transgenic poultry. The idea is to coat sperm with the desired gene (DNA) prior to insemination. Using sperm to transmit DNA is an appealing strategy because of the ease in obtaining sperm and their ability to penetrate the ovum. However, although DNA may be readily attached to sperm, successful integration of transferred genetic material by this method has not been reported (Freeman and Bumstead, 1987; Salter and Crittenden, 1988, personal communication).

Several strategies designed to lead to more efficient methods of gene transfer, through manipulating early embryos and embryonic cells, are worth noting. One such method recently developed is the *in vitro* culture of chicken embryos isolated from the magnum of the oviduct (Perry, 1988). This method may make it possible to insert new genes by microinjecting DNA or by infecting by retroviral vector at the one-celled stage of embryonic development. It also may reduce the mosaicism associated with introducing genes at later stages of development. The main disadvantage of this procedure is the low production rate (three percent) of healthy chicks which may limit its usefulness.

Another method of gene transfer is the production of chimeras by transferring cells isolated from one blastoderm to another by microinjection (Petitte and Etches, 1988). Similarly, donor primordial germ cells may be isolated from the germinal crescent and transferred to early embryos where they colonize the host gonad (Shuman, 1981; Shuman and Shoffner, 1982; Wentworth, 1988, personal communication). Although it has not been determined at what frequency the donor cells will populate the germ line, it may be possible to transfer genes to donor cells prior to chimera formation. The main limitation of this procedure is the inability to select donor cells that contain transferred genes. Developing embryonic stem cell lines may overcome this limitation. In the mouse, embryonic stem cells may be grown in culture and reintroduced into blastocysts where they colonize the embryo including the germ line (Bradley et al., 1984). Mice have been produced from selected embryonic stem cell clones that contain genes inserted by DNA transfection or retroviral infection (Kuehn et al., 1987). To date, however, embryonic stem cells have been produced only in the mouse. For this method to be applicable in commercial poultry breeding, it may be necessary to develop many stem cell lines with suitable genetic backgrounds.

APPLYING GENETIC ENGINEERING TECHNOLOGY

Several steps are necessary in developing, testing, and producing transgenic poultry (Figure 24.2). First, the development phase begins with selecting a genetic characteristic to be modified. Genes to be transferred must be additive or dominant in action to produce an effect on the hemizygote. Then molecular genetic technology is used to identify and isolate the gene and regulatory sequence that will give the desired genetic characteristic. Characteristics may be modified by either inserting genes or by inhibiting gene expression

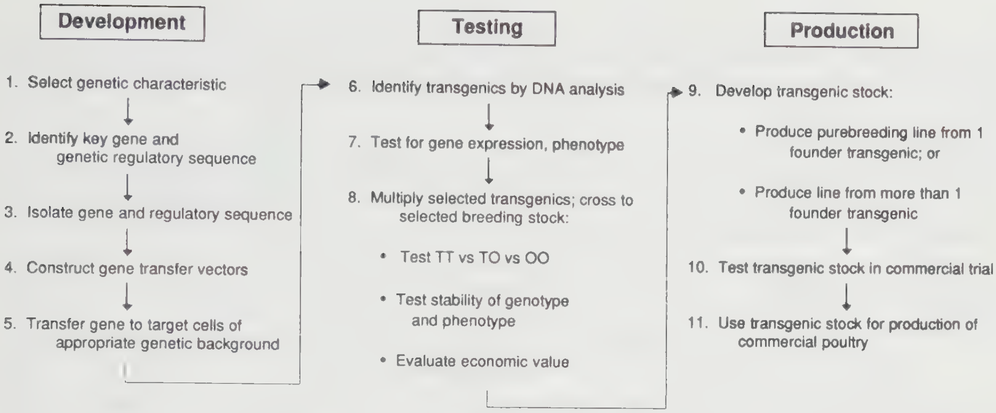
Development
Testing
Production
1. Select genetic characteristic
2. Identify key gene and genetic regulatory sequence
3. Isolate gene and regulatory sequence
4. Construct gene transfer vectors
5. Transfer gene to target cells of appropriate genetic background
6. Identify transgenics by DNA analysis
7. Test for gene expression, phenotype
8. Multiply selected transgenics; cross to selected breeding stock:
• Test TT vs TO vs OO
• Test stability of genotype and phenotype
• Evaluate economic value
9. Develop transgenic stock:
• Produce purebreeding line from 1 founder transgenic; or
• Produce line from more than 1 founder transgenic
10. Test transgenic stock in commercial trial
11. Use transgenic stock for production of commercial poultry

Figure 24.2. Steps in the development, testing, and production of transgenic poultry. TT = transgenic homozygote; TO = transgenic heterozygote; OO = nontransgenic.

by inserting sequences to produce antisense RNA (Izant and Weintraub, 1985). Regulatory sequences may rely on control by host cells; however, it may be necessary to devise sequences that can be controlled externally. If a retrovirus vector is used for gene transfer, the next step is inserting the gene into the vector. Vector virus may then be used to transfer the gene to an early embryo or other target cell with the appropriate genetic background.

In most cases, genes should be inserted directly into the breeding stock in which the new characteristic is desired. This is important because inserted genes may behave differently depending on the genetic background. Directly inserting genes into the breeding stock also reduces genetic lag, the time required to develop a transgenic line. However, if possible, a duplicate nontransgenic line should be maintained in a parallel selection scheme. This will reduce risk and serve as a control population in determining the economic value of the transgenic line.

Next, the testing step begins by identifying birds carrying the foreign gene. DNA analysis is used to screen potential transgenic birds to establish the presence of the transferred gene. Birds carrying the transferred gene are then tested for expression and the phenotype produced. Selected transgenic birds are then multiplied by crossing to selected breeding stock and examined for stability of the genotype and phenotype. It is particularly important to assess the viability and fertility of homozygotes and to compare their performance to hemizygotes and nontransgenic birds. Reduced viability and fertility have been found in other transgenic animals, and are often caused by mutagenesis of a host gene due to insertion of the transferred gene (Palmiter and Brinster, 1986).

Thirdly, two approaches exist for producing transgenic breeding stock. The simplest and most straightforward is to produce a purebreeding line from one founder transgenic bird. This may be accomplished by first crossing selected transgenic birds to selected nontransgenic birds (whose selection is based on economic value or index) to reduce genetic lag, and then crossing selected hemizygous birds to produce homozygotes as shown in Table 24.1 (Smith et al., 1987). However, the main disadvantages of this strategy are the inbreeding incurred because all members of the line descend from one common ancestor, and the likelihood of having to develop several transgenic lines to identify the best 'nick' upon crossing.

Table 24.1. Strategy for developing purebreeding transgenic lines for commercial poultry production, research, or other applications. (Modified from Smith et al., 1987).

Generation	Operation	Birds
0	Progeny test Mate	Founder transgenic[1] (TO), nontransgenic (OO) Selected[2] founder (TO) x selected nontransgenic (OO)
		↓
1	Progeny test Mate	Hemizygous progeny (TO) Selected TO x TO[3]
		↓
2	Progeny and lethality test Mate	Homozygous progeny (TT, inbreeding six percent) Selected TT x TT
		↓
3-5[4]	Begin commercial trial	Selected TT x commercial line
4+	Begin commercial production	Selected TT x commercial line

[1] Males preferred to increase number of progeny obtained.

[2] Selection based on economic value or index.

[3] May mate selected TO x selected OO to reduce inbreeding to three percent; add one generation to development time.

[4] Length of commercial trial may depend on gene inserted and other factors.

A second approach is to produce a line that contains more than one transgenic founder. Here, selection would derive from economic value, and would gradually fix genes with the most useful effects and eliminate those less useful or deleterious. This method has the advantage that it reduces inbreeding and favors the best genotype combinations; however, it creates a much more complex task for identifying and analyzing the effects of transferred genes and may delay the time to fixation.

Once a transgenic line is produced, it must be tested in commercial trials prior to being used in commercial production. Perhaps, the foremost impediment to commercial production will be a decrease in genetic gain compared to selected nontransgenic lines. This is due to the time required to produce the line, which is estimated to be several generations. Thus, the economic benefit of the transferred gene would need to be large enough to make developing the transgenic line worthwhile. For example, in the poultry industry the rate of genetic change using selection for growth rate has been estimated to be 6.5 percent of the mean/year, and to be 1.7 percent for egg production (Smith, 1984). Therefore, the effect of the transferred gene on the genetic characteristics to be modified, and the overall effect on the economic value, must be greater than the 'cost' of producing the line.

Gene transfer and genetic engineering potentially may be applied to genetically improving poultry, to basic research, and to producing pharmaceuticals and other biologically important proteins. The main objective of genetic improvement is to produce breeding stock that makes poultry production more efficient. Research to date has focused on growth enhancement, as in transfer of growth hormone genes (Souza et al., 1984), and on disease resistance, as in transfer of the ALV envelope gene (Salter and Crittenden, 1988). Other areas that appeal to poultry breeders are general enhancing of the immune system and reproductive function, altering nutritional requirements, reducing fat and cholesterol in meat and eggs, controlling sex, identifying the sex at hatching, and improving egg and meat quality.

Further, gene transfer may be viewed as an important new tool for basic research. This technology permits the study of cloned genes in the intact bird, and will likely be an important step in the process of identifying and testing genes to be used for improving poultry.

Finally, gene transfer may also create new uses for poultry. For example, it may be possible to employ the avian oviduct as a biological production system. This would be accomplished by transferring genes that code for proteins such as human blood clotting factors, or genes that code for industrial enzymes important to human and animal medicine and other industries. Using a biological system of this kind stems from the knowledge that some proteins require post-translational processing that cannot be carried out in bacterial systems. The desired protein gene could be fused with albumen protein genes and ultimately extracted from the egg.

Using molecular markers to identify breeding stock. Another technology evolved from molecular genetics that promises to benefit poultry breeding involves using molecular genetic markers to aid in evaluating and testing breeding stock for quantitative traits. Several factors reduce the effectiveness of selecting for economically important traits. The most troubling is that the phenotype may not accurately reflect the genotype, particularly for traits with low heritability. Another problem is that some traits are expressed late in life or only in one sex. Also, measuring some traits such as carcass yield requires slaughtering of the bird.

Genetic markers potentially could be used to identify a quantitative trait of economic value, or a quantitative trait locus (QTL). The objective is to correlate quantitative traits to the analysis of DNA. Molecular methods that examine DNA are advantageous in that sex, age, or tissue type do not restrict them. One proposed method is using restriction fragment length polymorphisms (RFLPs) (Beckman and Soller, 1983; Soller and Beckman, 1986). RFLPs are a class of genetic markers formed by point mutations or other DNA changes, and revealed by probing restriction enzyme-digested DNA with cloned genes. Theoretically, it is possible for RFLPs to identify QTL directly or to identify linkage between the RFLP locus and the QTL locus. It has been suggested that linkage tests would require examining several hundred RFLP loci in better than 1,000 birds in a segregating population (Beckman and Soller, 1983; Soller and Beckman, 1983). In practice, the small number of available markers and low levels of polymorphism (Beckman et al., 1986) has limited using RFLPs.

For these reasons, attention has begun to focus on the use of DNA fingerprints' as genetic markers in poultry breeding. These are another class of DNA markers that can be used to identify individual birds, determine the sire and dam, and facilitate linkage analysis (Gill et al., 1985; Jeffreys et al., 1985, 1986). DNA fingerprinting uses human minisatellite probes that crosshybridize to avian and other animal minisatellites (Burke and Bruford, 1987; Jeffreys et al., 1987). Consisting of tandemly repeated core sequences, these probes can detect high levels of polymorphism in chickens and other birds (Burke and Bruford, 1987). Future studies should reveal whether specific fingerprints can be correlated with traits such as growth or egg production.

POLITICAL AND SOCIAL ISSUES CONCERNING GENETIC ENGINEERING

Several political and social issues have been raised with respect to genetic engineering of poultry and other animals. One concern that may dramatically affect the poultry industry is animal patents. Artificial insertion of genes has opened the door for the patenting of animals in the U.S.A. and other countries. However, this concept has lead to pending legislation to limit the patenting of animals and to exempt farmers from paying royalties on patented animals. Nevertheless, it appears that poultry breeders in the future may seek patent protection for transgenic lines.

Further, because this technology allows poultry breeders and others to make genetic changes not thought possible until recently, many feel that molecular geneticists are 'playing God'. In addition, others fear that gene transfer will lead to unfair manipulation of animals, or worse, that microbes or animals produced by this new technology will become genetic monsters. Also, consumers and others fear that transferring foreign genes may lead to unsafe food products.

It will be the responsibility of the scientific community to prove the safety and benefit of this new technology and to overcome negative public opinion. Scientists must make it clear that developing and testing transgenic animals will follow strict guidelines which have been adopted for recombinant DNA research. Also, the public must know that government agencies will take the lead role in regulating and approving gene transfer methods and transgenic animals, as well as examining the safety and efficacy of transferred genes.

CONCLUDING REMARKS

Gene transfer, genetic engineering, and DNA fingerprinting are powerful new tools for the poultry breeder. There exists now the potential to produce poultry with new genetic characteristics. Yet it is probably incorrect to believe that this technology will be used to produce a 'superchicken'. It is more likely that over time these techniques will be incorporated into current poultry breeding practices to solve specific problems and to create new products. As with any new technology, research, development, and application will require a stepwise approach that includes much careful testing and evaluating. Nonetheless, it appears that poultry breeding is on the threshold of a new era.

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Part IV. QUANTITATIVE GENETICS AND SELECTION

Chapter 25. GENETICS OF GROWTH AND MEAT PRODUCTION IN CHICKENS

J.R. Chambers

Growth, defined simply as an increase in size, consists in animals of increases not only in cell size (hypertrophy), but also in cell number (hyperplasia), and in extracellular fluid (Widdowson, 1980). During the early embryonic development of the chicken, growth is the result primarily of hyperplasia; after hatching, growth of most tissues is largely, in some cases totally, due to hypertrophy. Animal growth has been defined by Moran (1977) as the sum of the growths of the component parts of the carcass, i.e. meat, bone, and skin. These parts not only differ in their rates of growth as age advances, but they are dependent upon levels of nutrition as well.

Growth is a very dynamic process. The theory of priority of partition of nutrients among developing tissues according to their metabolic rate in animals was described by Hammond (1947). Due to the different priorities of nervous, reproductive, skeletal, muscular, and adipose tissues (listed in declining order of priority), the level of nutrition not only influences the development of various tissues of the body but also causes greater variability in the development of some tissues than of others. This in turn influences meat composition. Jull and Titus (1928) reported that growth increments for successive kg of feed were reduced by about eight percent. Fox and Bohren (1954) confirmed this trend with their observation of a "negative regression of efficiency on average weekly body weight". They also demonstrated that faster compared with slower growing chickens were more efficient. In growing chicks, the energy costs of production in both net energy and heat increment change as the broiler grows due to the changing composition of the rapid body weight gain (Luiting 1987). In a review of energetic efficiency of growth in animals, Webster (1980) stated the following:

1. Metabolizable energy, the physiological fuel for the body, is partitioned between heat production and energy retained in the growing body principally as protein and fat.
2. During uninterrupted growth, body weight and parameters such as lean mass, body fat content and metabolic rate increase from conception along sigmoid curves to an asymptotic value at maturity.
3. During growth, metabolizable energy exceeds heat production. These values converge as maturity is approached.
4. Overall efficiency of energy retention peaks early in life and declines rapidly thereafter. As the animal matures, the ratio of fat to protein increases. Energy contents of lean meat and fat are about 4.8 and 39 kJ/g respectively. Thus the ratio of weight gain to retained energy declines throughout growth.

5. Feed utilization efficiency, reflecting both retained energy per unit of metabolizable energy and energy content of body gains, is relatively constant during the first third of growth and then begins to decline rapidly.
6. Maturity dominates the energetic efficiency of growth. Possible biochemical manipulations of the energetics of growth can only be assessed when animals are compared at the same stage of maturity.

These points add emphasis to the dynamic aspect of processes contributing to growth. Hence, the comparison of growing broilers with regard to traits other than growth must be performed at similar body weight or weight gain if accuracy is important.

The preceding material forms a basis for the understanding of such chicken meat production traits as growth, meat composition and yield, and feed consumption and efficiency. Comprehension of the dynamic aspects of this material will aid in the understanding of discussion to follow.

This chapter discusses results of research involving the genetics of growth, meat production, and related traits of chickens developed and/or used specifically for meat production. Research on Leghorns and other light breeds will be excluded unless they are a part of a study of meat breeds. The reader is advised to consult Chapters 26, 27 and 28 for relevant information from other meat-producing species of poultry.

Estimation of genetic parameters. Numerous biometrical procedures exist for the estimation of genetic parameters such as heritability and genetic correlation, and their reliability. Lush (1940) defined heritability as the fraction of the observed variance caused by differences in heredity. Estimation of heritability is based on likeness among relatives. These estimates are valid providing that the mating system is random and environmental deviations among groups of relatives are not correlated. Deviations from these conditions will distort estimates of heritability. Lush (1940) also distinguished between heritability in the broad sense, the functioning of the genotype as a whole, versus the narrow sense, the average effects of the constituent genes. The former includes variation due to additive effects of genes, dominance effects, epistatic effects, and effects of interaction between genes and the environment; the latter includes variation due to additive effects of genes but it includes only fractions of the dominance and epistatic effects. Heritabilities in the narrow sense are of most interest to breeders because it is the gene, not the genotype, that is the hereditary unit of transmission.

There are two major procedures for estimating heritabilities: using variance components analyses of data from either nested (hierarchical) mating designs (King and Henderson, 1954) or factorial (in special cases, diallel) mating designs (Comstock and Robinson, 1948; Goodman and Jaap, 1960), and secondly, using regression of offspring on parent (Lush, 1940; Kempthorne and Tandon, 1953). The methods of estimation of heritability are usually intermediate in terms of broad versus narrow heritability; however, some methods are better estimators of heritability in the narrow sense. Becker (1984) has outlined the types of genetic effects expected to contribute to heritability estimates based on sire, dam, or sire and dam components of variance from either nested or factorial mating designs, as well as those based on the sire-dam interaction variance component from factorial mating designs. Factorial designs make it possible to distinguish among additive genetic, nonadditive genetic, and sex-linked genetic and maternal effects better than nested designs. Lush (1940) expressed a preference for the regression analysis method when calculated on an intrasire basis. His reasons were: no dominance effects were included; deviations from random mating and selection of dams had less disruptive influences on estimates; and multiplication factors for intraclass correlations and regressions of offspring on parent are four and two, respectively, causing errors in the former to be larger than in the latter. The relative efficiencies of various methods of calculating regressions of offspring on parent have been examined by Kempthorne and Tandon (1953) and by Bohren et al. (1961). The calculation of confidence limits has been described for heritabilities based on variance components (Graybill et al., 1956; Graybill and Robertson, 1957) and on regression of offspring on parent (Kempthorne and Tandon, 1953).

Genetic correlations, like heritabilities, can be estimated for pairs of traits using either variance and covariance components (Snedecor, 1940) or the regressions of one of the pair of traits of offspring on the other trait of parents, and vice versa (Hazel, 1943). For the former, calculations have been described for approximate sampling variances by Osborne and Paterson (1952) and Robertson (1959a), and for exact sampling variances for estimates based on the sire intraclass correlation (Bogyo and Becker, 1963). For parent-offspring correlations, Reeves (1955) described procedures to determine the variance of the coefficient.

Calculations required to determine realized heritabilities using selection response and cumulative selection differentials have been demonstrated by Dickerson and Grimes (1947).

Procedures for determining optimum group size for progeny testing and family selection, and for experimental design in evaluation of genetic parameters, have been presented by Robertson (1957, 1959b).

For the estimation of heritabilities, it is obvious that studies will and do vary in the procedures used, the strains or breeds of chickens used, the sizes of the parental and filial flocks, the sexes contributing to each estimate, and the number of generations contributing to each estimate. To aid in summarizing published heritability estimates for this chapter, populations have been rated with regard to the numbers of parents and progeny. Populations were considered to be small if there were fewer than 20 sires, 100 dams and 1000 progeny, to be large if there were at least 100 sires, 1000 dams and 10,000 progeny, and to be medium if values were intermediate to these extremes. In an attempt to make studies similar in their basis of estimation of heritabilities, estimates for each sex, for each replicate line, or for different generations have been averaged for each study to obtain a common basis of comparison.

GROWTH

Measurement Of Growth

Growth is a complex physiological process that exists from conception until maturity in animals. Consequently accurate measurement of the entire growth phase cannot be performed easily. Poultry breeders tend to use simplified and practical measures which are described below to evaluate growth of chickens.

Weight. Body weight at a specific age is probably the most frequently used indicator of growth. It is attractive because it is relatively easy to measure. This measure is a good indicator of accumulated growth to the age of measurement. It gives no indication of differences in growth rate during component intervals up to the measurement age. Due to high correlations between different body weights, a body weight is usually a fair indicator of subsequent growth rate.

Weight gain. Body weight gain during a given interval may be used to indicate average growth rate during the interval measured. It tends to be highly positively correlated with other weights and gains during growth. It requires two body weight measurements and subsequent calculation. Body weight gain gives little indication of any changes in growth rate during the interval of measurement. This trait is often measured as part of feed efficiency (or the inverse, feed conversion ratio).

Growth curve parameters. Appropriate mathematical functions have the potential to concisely and biologically represent the entire growth phase of the chicken. Graphic illustration of these functions gives rise to growth curves. These curves are universal sigmoid curves which can define the growth rates, not only of chickens, but also of animals and plants. Growth curves have four characteristics: an accelerating growth phase following hatch, a point of inflection coincident with maximum growth rate, a decelerating growth phase, and a limiting mature weight which is approached asymptotically. Baker (1944) has listed a number of factors influencing growth. These include heredity, nutrition, management, other environmental conditions, random sampling variation, and nonrandom selection of chickens to be studied. Wilson (1977) recommended use of growth models that include descriptions of feed intake for more accurate interpretation of growth studies. Growth curves, which fit a series of body weights well, can provide a concise summary of the weight responses required to define accurately and thoroughly in terms of parameters, either parts of or the entire growth response. Constants of growth curves have been used not only to quantify but also to qualify differences in growth curves between sexes, breeds, and strains (Lerner and Asmundson, 1938). Unfortunately, body weights at several stages of growth are necessary for appropriate growth curve selection. Calculations are also required for fitting and determining the parameters of the curve. In addition, it is often necessary in applied poultry breeding to evaluate growth during juvenile stages to permit choice of actions long before the chicken achieves maturity. Lerner and Asmundson (1932) adopted a formula based on weights of the growing chicken at two ages to represent growth. They demonstrated the feasibility of using this formula to reduce growth rates to arbitrary values in order to compare growth of Light Sussex, Ancona and hybrid progeny chickens at juvenile stages. The use of body weights and weight gains to indicate growth rate represents the ultimate in simplification of growth curves. The objections mentioned above have deterred the use of growth curves in spite of early recognition of their benefits.

Breed And Strain Differences In Growth

Early investigators of the inheritance of growth attempted to determine the importance of heredity in growth and the nature of the genetic factors involved. Such attempts were made by studying the performance of chickens of various breed or strain crosses.

Several early studies provided evidence of inheritance of growth rate. Twelve-week body weight and shank length showed genetic differences. Both were highly positively correlated, but neither was correlated with 8-12 week gain, and results indicated that growth inheritance is complex (Lerner and Asmundson, 1932). Growth rates to 24 weeks of age of White Leghorn and Barred Plymouth Rock chickens were compared (Asmundson and Lerner, 1934). Rocks grew more rapidly than Leghorns to 16 weeks, but the two breeds did not differ thereafter. Differences in growth rate were greatest for intervals between two and 16 weeks of age with little difference thereafter. Data from a crossbreeding study reported by Warren were used by Lerner and Asmundson (1938) to study genetic growth constants. Constants were used to confirm evidence of sex-linked effects on growth rates reported by Warren, to demonstrate breed and strain differences in growth, and to demonstrate that early differences in growth rate reflected differences in adult weight. Lerner (1937) presented evidence of heterogonic growth in a comparison of Plymouth Rock and Minorca chickens with bantams. The two standard breeds and their crosses had, within sex, similar patterns of growth for pectoralis major muscles and for lengths of leg bones. The rates of growth for parts exceeded the rate for body weight. Bantams had leg bones that grew at a slower rate than body weight, and breast muscles that grew at the same rate as body weight.

From a common population of Barred Plymouth Rock chickens, divergent selection for body weight at 8-9 weeks of age was practiced in both sexes in the first generation and only in males in the two subsequent generations (Schnetzler, 1936). The first and second generation progenies of the two lines differed significantly in body weights at eight weeks and at maturity giving evidence that differences in growth rate are inherited.

Studies of purebreds, their F_1 and later crosses, and their backcrosses were used to evaluate the nature of genetic factors determining growth rate. Unfortunately limited numbers of chickens, either as parents or in specific crosses or subclasses, together with poor control of environmental influences, limited the potential of some of these studies. Lerner (1937) outlined the following lines of evidence from crossbreeding studies supporting inheritance of growth: greater variability in F_2 as opposed to F_1 populations, genetic distinctness of F_3 progenies in contrast to performance of F_2 parents (there is a general lack of distinctness of F_2 progenies of F_1 parents), linkage of some genes for quantitative characters, evidence that several chromosomes may have an influence on a specific trait, and occurrence of heterosis. Greater variability of growth in F_2 as opposed to F_1 generations was demonstrated by Punnett and Bailey (1914), Jull and Quinn (1931), Waters (1931), Maw (1935), and Godfrey (1953). Genetic distinctness of F_3 progenies was shown by Punnett and Bailey (1914), with further evidence of genetic segregation from Waters (1931). A sex-linked gene influencing growth was reported by Lerner and Asmundson (1938 from Warren's data), Maw (1935) and Godfrey (1953). The latter two studies indicated that many other genes also influenced growth; it can be concluded that these were autosomal. Evidence of linkage of qualitative genes with genes influencing growth is not plentiful. Godfrey (1953) located the sex-linked gene associated with growth midway between the *S* and *K* loci. Heterosis influencing growth was reported by May (1925), with support from Waters (1931), for growth to three or four months. In contrast, Jull and Quinn (1931) reported negative heterosis for growth which was attributed to dominance for small body size in Rose Comb Black bantams which had been crossed to Barred Plymouth Rocks. The earlier studies estimated that there were two to four major genes, whereas later studies estimated there were many (15+) genes determining growth rate.

The preceding information may not be definitive in all aspects. However, it does reveal that some differences in growth rate are hereditary, the genetic basis is polygenic, at least one growth gene is sex-linked, heterosis may exist especially for earlier body weights, and growth rates may be changed by genetic selection.

Heritability Of Growth

Excellent summaries of heritabilities of growth rate have been presented by Siegel (1962b) and Kinney (1969). Additional estimates have been published since these reports. These heritability estimates permit evaluation of not only the magnitude but also of various types of heritabilities. Heritabilities of measures of growth such as body weights and gains will be discussed by type as follows: estimates of heritability based on either variance components or regression coefficients; realized heritabilities based on total response to selection or average response per generation of selection. Where possible, the various estimated and/or realized heritabilities will be compared.

Heritability estimates for body weights and for weight gains, based on variance components from nested mating designs, are summarized in Tables 25.1 and 25.2, respectively. Estimates based on sire components are lowest (.4-.6), whereas estimates based on dam components are highest (.7). Estimates based on both sire and dam components are intermediate (.5-.6). The above situation applies not only to body weights but also to weight gains. Moreover, these measures of growth both have moderate to high heritabilities.

Data from pedigreed chickens produced from a factorial or diallel mating design permit the geneticist to partition the dam component of variance obtained from analyses of data from nested mating designs into dam and sire-dam interaction variance components. This dam variance component is similar in genetic expectation to the sire variance component, except that the dam component includes maternal effects and differs in additive sex-linked genetic effects (Becker, 1984). The latter difference depends upon the sex of the offspring. The sire-dam interaction variance component consists of portions of the nonadditive genetic effects due to dominance, epistasis, and dominance-epistatic interactions. Nonadditive genetic effects and maternal and additive sex-linked genetic effects can then be estimated by using these measures. Heritability estimates for body weights and gains based on variance components from factorial or diallel mating designs are presented in Table 25.3. The heritabilities based on sire, dam, sire and dam variances, or on additive genetic variance differed little (.44 to .48). Hence, nonadditive genetic, sex-linked genetic, and maternal effects, which are normally small and often of questionable significance, are responsible for the increase in size of heritability estimates containing dam variance components in studies using nested mating designs. Individually these effects are small; however, their combined effects when multiplied by factors of two to four are quite significant.

Heritability estimates based on data from both nested and factorial mating designs vary greatly not only among but also within studies (<0 to >1). Sampling variation associated with relatively small numbers of observations per estimate appears to be responsible. However, differences among genetic groups and environments will be contributing factors as well. In addition, other factors which give rise to either large sire and small dam components or vice versa appear to be active. These factors probably include assortative mating, either intended or otherwise, and persistent deviations from average variation among the females mated to each male.

Realized heritabilities for body weight based on response to selection are summarized in Table 25.4. Values are lower (.3-.4) than those based on either sire and dam or dam variance components presented in earlier tables. Realized heritabilities based on total selection response utilize information from only the final generation. When selection has been practiced for several generations, such estimates will be more prone to environmental error peculiar to the final generation than will estimates obtained by regression of selection response on selection differential for all generations. It is not clear why the heritabilities based on response per generation should be higher than those based on total selection response (.4 vs. .3). This phenomenon is probably due to sampling and the relatively small number of studies contributing estimates of total selection response.

There have been several reports of body weight heritability estimates derived from regression of offspring on parent. Often the same data have been used to estimate heritabilities from variance components or from response to selection. Heritabilities estimated using variance components and regression of offspring on parent can be compared

Table 25.1. Heritability estimates for body weight based on variance components from nested mating designs.

Type of estimate[1]	Average	No. of values	No. of studies
S, all studies	.41	53	23
D, all studies	.70	39	19
S+D, all studies	.54	26	14
S, studies with S and D	.36	26	10
D, studies with S and D	.71	26	10
S, studies with S, D and S+D	.58	13	9
D, studies with S, D and S+D	.70	13	9
S+D, studies with S, D and S+D	.64	13	9

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age	Basis of estimate[1]
Lerner et al. (1947)	NH	S	1	12 wk	S, D, S+D
El-Ibiary and Shaffner (1951)	NH	S	1	2, 6, 8, 10 wk	S, S+D
Hurry and Nordskog (1953)	NH, BPR	S	1	8 wk	S+D
Kan et al. (1958)	?	S-M	1	4, 9 wk	3S+D
Thomas et al. (1958)	NH	M	1	4, 6, 8, 10 wk	S, D
Siegel and Essary (1959)	WPR	S-M	2	59 d	S, D, S+D
Siegel and Siegel (1960)	WPR	S-M	1	62 d	S+D
Jaap (1962)	?	S	2	8 wk	S, D
Friars et al. (1962)	WPR	M	7, 9	8 wk	S, D, S+D
Moyer et al. (1962)	CxNH	M	2	4, 6, 8 wk	S, D
Siegel (1963a)	WPR	M	5	4, 24, 38 wk	S+D
Siegel (1963b)	WPR	M	5	4, 24, 38 wk	S+D
Ricard and Rouvier (1967)	BrPi	S	1	11 wk	S, D, S+D
Merritt (1968)	OMC	L	5	6, 9, 21, 44 wk	S, D
Mérat and Prod'homme (1969)	Synth	M	1	8 wk	S, D, S+D
Ricard and Rouvier (1969)	C	S	1	59 d	S, D
Goodman (1973)	ACRB	M	5	?	S, D, S+D
Ricard (1974)	Synth	M	4	80 d	S, D
Ricard (1975)	BrPi	S	3	8 wk	S, D
Becker et al. (1979)	C	M	1	7 wk	S, D
Pym and Nicholls (1979)	Synth	M	1	5, 9 wk	S, D, S+D
Ayoub et al. (1980)	WPR, LS, x	M	2	8 wk	S, D, S+D
Marks (1980)	ACRB	M	2	6, 8 wk	S
Becker et al. (1984)	WR	M	1	50 d	S, D, S+D
Chambers et al. (1984)	C, WR	M	1	4, 6, 7 wk	S
Cahaner et al. (1985)	WR	S	1	7, 9 wk	S
Cahaner and Nitsan (1985)	WR	S	1	9 wk	S+D
Leenstra et al. (1986)	C	M	1	6 wk	S, D
Leenstra and Pit (1988)	C	M	1	6 wk	S, D

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[1] S = sire; D = dam; S+D = sire and dam.

[2] ACRB = Athens-Canadian randombred; BPR = Barred Plymouth Rock; BrPi = Bresse Pile; C = Cornish; LS = Light Sussex; NH = New Hampshire; OMC = Ottawa Meat Control; Synth = synthetic; WPR = White Plymouth Rock; WR = White Rock; WW = White Wyandotte; x = crossbred.

[3] Popn. size = population size; S = small; M = medium; L = large.

[4] Gen. = generations of measurement.

Table 25.2. Heritability estimates for weight gain based on variance components from nested mating designs.

Type of estimate[1]	Average	No. of values	No. of studies
S, all studies	.50	8	5
D, all studies	.70	7	4
S+D, all studies	.64	3	2
S, studies with S and D	.42	4	2
D, studies with S and D	.68	4	2
S, studies with S, D and S+D	.60	3	2
D, studies with S, D and S+D	.72	3	2
S+D, studies with S, D and S+D	.64	3	2

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age	Basis of estimate[1]
Kan et al. (1958)	?	S-M	1	4-9 wk	3S+D
Thomas et al. (1958)	NH	M	1	4-6, 6-8, 8-10 wk	S, D
Smith and Goodman (1971)	ACRB	M	1	12, 50 d	S, D, S+D
Pym and Nicholls (1979)	Synth	M	1	5-9 wk	S, D, S+D
Chambers et al. (1984)	C, WR	M	1	4-7 wk	S
Leenstra et al. (1986)	C	M	1	3-6 wk	S, D

[1] S = sire; D = dam; S+D = sire and dam.
[2] ACRB = Athens-Canadian randombred; C = Cornish; NH = New Hampshire; Synth = synthetic; WR = White Rock; ? = unknown.
[3] Popn. size = population size; S = small; M = medium; L = large.
[4] Gen. = generations of measurement.

from the studies summarized in Table 25.5. Again, the estimates based on the sire variance component are smallest (.4), those based on the dam variance component are largest (.6), and those based on sire and dam variance components are intermediate (.5). Estimates based on the regression of performance of offspring on parent are similar to those based on the sire component of variance (.4). The regression estimates are not only smaller than some of the other types of estimates, but they also seem to be less variable.

Studies which include combinations of heritability estimates based on variance components and/or regression of offspring on parent with those based on selection response are summarized in Table 25.6. As in earlier tables, estimates based on sire variance components are smaller than those based on sire and dam variance components, which in turn are smaller than those based on dam components (.4 vs. .5 vs. .6, respectively). Estimates based on regression of offspring on parent are lower than all other estimates (.3 - .37), but are close to estimates based on the sire component of variance. Realized heritabilities are higher (.4-.6), tending to exceed estimates based on sire variance components, and in some instances being equal to those estimates based on sire and dam variance components.

From Tables 25.4, 25.5, and 25.6, it becomes apparent that there have been numerous selection studies focusing on growth as reflected by body weight or gain at specific ages in meat-type chickens. Few studies have extended beyond 12 generations of selection.

Table 25.3. Estimates of heritability and nonadditive genetic, sex-linked genetic, and maternal variances for growth traits based on factorial (diallel) mating designs.

Type of estimate[1]	Average	No. of values	No. of studies
S, all studies	.44	8	4
D, all studies	.46	8	4
S+D, all studies	.48	8	3
Additive variance, all studies	.45	3	2
S, studies with S and D	.44	8	4
D, studies with S and D	.46	8	4
S, studies with S, D and S+D	.62	1	1
D, studies with S, D and S+D	.28	1	1
S+D, studies with S, D and S+D	.45	1	1

Nonadditive genetic	In ten studies, effects were either nonsignificant or were small when significant.
Sex-linked genetic	In three studies, effects were usually nonsignificant and small.
Maternal	In four studies, effects were either nonsignificant or were small when significant.

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age (wks)	Basis of estimate[1]
Martin et al. (1953)	RIR	S-M	1	3, 6, 9	S+D, NA, X, M
Brunson et al. (1956)	NH, SO, x	M	1	10	S, D, S+D, NA, X, M
Goodman et al. (1957)	NH	?	1	8	S, D, NA
Goodman and Jaap (1960)	NH	M	1	8	S, D, NA
Smith and Jaap (1957)	RB	S-M	1	8, 16, 24	S+D, NA, X, M
Jaap and Smith (1959)	?	M	3	8	A, NA, M
Jaap et al. (1962)	OWG	M?	5	8, 16, 24	S, D, NA
Mahmoud et al. (1965)	NH	S-M	?	8	A, NA

[1] S = sire; D = dam; S+D = sire and dam, NA = nonadditive genetic; X = sex-linked genetic; M = maternal variance.
[2] NH = New Hampshire; OWG = Ohio White Gold; RB = randombred; RIR = Rhode Island Red; SO = Silver Oklabar; x = crosses; ? = not specified.
[3] Popn. size = population size; S = small; M = medium; L = large; ? = not specified.
[4] Gen. = generations of measurement.

Two exceptions are those reported by Maloney et al. (1963a, 1967) at Oklahoma and by Siegel (1978), Barbato et al. (1983) and Dunnington and Siegel (1985) at Virginia Tech. In the Oklahoma study, flocks of Silver Oklabar chickens were selected divergently for 12-week body weight. The two lines differed by approximately 800 g after ten generations of selection. Results after 15 generations revealed greater response for high than for low body weight. In the Virginia study, flocks of White Plymouth Rock chickens were selected divergently for eight-week body weight. To 20 generations, the high weight line increased 26 and 20 g per generation for males and females, respectively (Siegel, 1978). Corresponding weight declines for the low weight line were 18 and 15 g, illustrating the asymmetric response. Maintenance of an unrelated control line permitted the distinction between selection response in the two lines. Realized heritabilities were higher for the high line (.26 and .23 vs. .17 and .17 for males and females, respectively) than for the low line. Results following seven additional generations of selection indicated that the realized heritability in the high line declined as selection progressed. It was actually negative (-.28) during the final eight generations, and accounted for a loss of 43 percent of the previous improvement (Dunnington and Siegel, 1985). In the low weight line, response and heritability were lowest (.14) during the second seven generations and highest (.49) during the final eight generations. The changes observed in the later generations were attributed, at least in part, to the high level of inbreeding (F=.39). This resulted from the limited number of parents selected and mated. Unfortunately the control line was unrelated and was maintained with more parents in the early generations. Otherwise it might have served to distinguish effects of selection and inbreeding. Relaxed selection lines established during the study were observed to regress toward control values. Breeders of generation 22 high and low lines were crossed to produce and compare performance of purebred and crossbred progeny (Barbato et al., 1983). Results indicated that the heritable variation of body weight

Table 25.4. Realized heritabilities and responses to selection for growth.

Type of estimate[1]	Average	No. of values	No. of studies
Total response, R_t	.31	4	2
Response per generation, R_b	.43	15	7
Response type unknown, $R_?$	.31	5	3
Responses, total and per gen., R_t	.31	4	2
Responses, total and per gen., R_b	.34	4	2

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age (wks)	Basis of estimate[1]
Godfrey and Williams (1952)	SO	M	2	6, 12	R_b
Godfrey and Goodman (1955)	SO	M	5	6, 12	$R_?$
Jaap and Smith (1959)	Br	M	3	8	-
Siegel (1962b)	WPR	S	4	8	R_t, R_b
Gyles and Thomas (1963)	WW	?	4	8	$R_?$
Maloney et al. (1963a)	SO	M	10	12	R_t
Carte and Siegel (1968)	WPR	S	4	8	R_t, R_b
Pym and Nicholls (1979)	Synth	M	5	5-9	R_t, R_b
Marks (1983)	ACRB	M	4	8	R_b
McPherson et al. (1985)	Br	?	8	7	R_b
Pym (1985)	Synth	M	10	5-9	$R_?$
Chambers (1987)	C	M	5	4	R_b

[1] R_b = regression by generation of accumulated selection response on accumulated selection differential; R_t = ratio in final generation of accumulated selection response on accumulated selection differential; $R_?$ = method not specified.

[2] ACRB = Athens-Canadian randombred; Br = broiler strain; C = Cornish; SO = Silver Oklabar; Synth = synthetic; WPR = White Plymouth Rock; WW = White Wyandotte.

[3] Popn. size = population size; S = small; M = medium; L = large; ? = unknown.

[4] Gen. = generations of measurement.

Table 25.5. Body weight heritability estimates based on variance components and regression of offspring on parent.

Type of estimate[1]	Average	No. of values	No. of studies
S, all studies	.41	8	3
D, all studies	.59	6	2
S+D, all studies	.47	16	7
b, all studies	.39	16	7
S, studies with S, S+D and b	.41	8	3
S+D, studies with S, S+D and b	.49	8	3
b, studies with S, S+D and b	.41	8	3
S, studies with S, D, S+D and b	.36	6	2
D, studies with S, D, S+D and b	.59	6	2
S+D, studies with S, D, S+D and b	.47	6	2
b, studies with S, D, S+D and b	.40	6	2

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age	Basis of estimate[1]
Kreuger et al. (1952)	Le, NH, RIR, PR	M	4	10 wk, housing	S+D, b
Dillard et al. (1953)	NH	M	4	8 or 12 wk	S+D, b
Wyatt (1954)	Le, NH, BPR, WPR, RIR, A, Ln, C, F	M	5	8 wk, housing, mature	S+D, b
Peeler et al. (1955)	RIR	M	2	10 wk, sex. mat.	S, S+D, b
Ghostley and Nordskog (1956)	6?	M	3	8 wk, mature	S+D, b
Godfrey and Goodman (1956)	NH, SO	M	1	9 wk	S, D, S+D, b
Goodman and Godfrey (1956)	NH, SO	M	4	9 wk, mature	S, D, S+D, b

[1] S = sire; D = dam; S+D = sire and dam; b = regression of offspring on parent.
[2] A = Australorp; BPR = Barred Plymouth Rock; C = Cornish; F = Fayoumi; Le = Leghorn; Ln = Langshan; NH = New Hampshire; PR = Plymouth Rock; RIR = Rhode Island Red; SO = Silver Oklabar; WPR = White Plymouth Rock; 6? = 6 unknown breeds.
[3] Popn. size = population size; S = small; M = medium; L = large.
[4] Gen. = generations of measurement.

in these lines of chickens was principally additive, with negligible portions due to heterosis.

Evidence of the dramatic genetic change in growth rate achieved by the broiler breeding industry in previous decades has been illustrated by Marks (1979) and by Chambers et al. (1981). In these studies, progeny from either the Ottawa Meat Control strain or a strain derived from it, the Athens-Canadian randombred, were used to represent broilers of 1955 in comparisons with commercial broilers obtained approximately two decades later. The Ottawa Meat Control strain was derived from four meat strains obtained in 1955. Both control strains had been maintained without artificial selection since their derivation. In both studies the modern broilers grew at least twice as fast as the experimental strains of broilers.

Table 25.6. Estimated and realized heritabilities for body weights.

Type of estimate[1]	Average	No. of values	No. of studies
S, all studies	.40	12	5
D, all studies	.63	5	3
S+D, all studies	.50	11	5
b, all studies	.32	11	6
R, all studies	.43	21	9
S, studies with S and R	.40	12	5
R, studies with S and R	.50	12	5
D, studies with D and R	.63	5	3
R, studies with D and R	.59	5	3
S+D, studies with S+D and R	.50	11	5
R, studies with S+D and R	.44	11	5
b, studies with b and R	.32	11	6
R, studies with b and R	.42	11	6
S+D, studies with S+D, b and R	.50	7	3
b, studies with S+D, b and R	.26	7	3
R, studies with S+D, b and R	.46	7	3
S, studies with S, D, b and R	.53	5	3
D, studies with S, D, b and R	.63	5	3
b, studies with S, D, b and R	.37	5	3
R, studies with S, D, b and R	.59	5	3
S, studies with all types	.56	4	2
D, studies with all types	.64	4	2
S+D, studies with all types	.60	4	2
b, studies with all types	.33	4	2
R, studies with all types	.61	4	2

Reference	Breed[2]	Popn.[3] size	Gen.[4]	Age	Basis of estimate[1]
Becker and Berg (1960)	WPR	M	1, 2	8 wk	S+D, R
Siegel (1962a)	WPR	S	5	8 wk	S, D, S+D, b, R
Kinney and Shoffner (1965, 1967)	Synth	M	4	8 wk	S, b, R
Ideta and Siegel (1966)	WPR	S	5	24, 38 wk	S+D, R
Merritt (1966); Merritt et al. (1966)	OMC	L	5	9 wk	S, D, b, R
Maloney et al. (1967)	SO	M	15	12 wk	b, R
Dev et al. (1969)	A, G, AG	M	5, 8, 11	8 wk	S+D, b, R
Marks and Britton (1978)	ACRB	S	2	6, 8 wk	S, R
Saxena and Mohapatra (1981)	WC, NH, WPR	M	3	10 wk	S, D, S+D, b, R

[1] S = sire; D = dam; S+D = sire and dam; b = regression of offspring on parent; R = response to selection.

[2] A = Ohio experimental strain; ACRB = Athens-Canadian randombred; AG = Ohio experimental strain; G = Ohio experimental strain; NH = New Hampshire; OMC = Ottawa Meat Control; SO = Silver Oklabar; Synth = synthetic; WC = White Cornish; WPR = White Plymouth Rock.

[3] Popn. size = population size; S = small; M = medium; L = large.

[4] Gen. = generations of measurement.

Relationships Among Various Measures Of Juvenile Growth

Many of the studies that reported heritabilities for measures of juvenile growth also reported genetic correlations among juvenile body weights and gains. Estimates of genetic correlations among these traits are listed in Table 25.7. Most of the correlations presented are very high (>.9), especially those among weights taken only a week apart. Lower values tend to involve either weights separated by several weeks or a weight and a subsequent gain interval. Correlations between weight and gain during the previous interval are larger (.9 vs. .6). The results of Aman and Becker (1983) suggest that correlations may be overestimated when the two traits are measured on the same chicken, possibly due to common nongenetic influences. If this were correct, the realized correlations listed in Table 25.8 could not be so large. They tend to match the values reported in Table 25.7.

Table 25.7. Genetic correlation (r_g) estimates between measures of juvenile growth.

Traits (Reference)	r_g	Breed[1]	Popn.[2] size	Gen.[3]	Basis of estimate[4]
3 & 6, 9 or 12 wk wt	.91, .83, .69	RIR	M	1?	S+D
6 & 9 or 12 wk wt	.97, .92				
9 & 12 wk wt	.98				
(Martin et al., 1953)					
8 & 16 wk wt	.77	OWG	M	1	S+D
(Jaap et al., 1962)					
6 & 9 wk wt	.95, .92, .94	OMC	L	6	S, D, b
(Merritt, 1966)					
5 & 9 wk wt	.80, .94, .86	Synth	M	1	S, D, S+D
5 wk wt & 5-9 wk gn	.57, .74, .64				
9 wk wt & 5-9 wk gn	.94, .92, .94				
(Pym and Nicholls, 1979)					
6 & 7 wk wt	.58	WR	M	1	S+D
6 & 7 wk carcass wt	.66				
(Aman and Becker, 1983)					
4 & 6 or 7 wk wt	1.03, .80	C, WR	M	1	S
6 & 7 wk wt	1.00				
Carcass wt & 4, 6 or 7 wk wt	.76, 1.00, .95				
4-7 wk gn & 4, 6 or 7 wk wt	.57, .83, .95				
4-7 wk gn & carcass wt	.91				
(Chambers et al., 1984)					
6 wk wt & 3-6 wk gn	.99	C	M	1	D
(Leenstra et al., 1986)					

[1] C = Cornish; OMC = Ottawa Meat Control; OWG = Ohio White Gold; RIR = Rhode Island Red; Synth = synthetic; WR = White Rock.

[2] Popn. size = population size; S = small; M = medium; L = large.

[3] Gen. = generations of measurement.

[4] S = sire; D = dam; S+D = sire and dam; b = regression of offspring on dam.

Table 25.8. Realized genetic correlations (r_g) between measures of juvenile growth.

Traits (Reference)	r_g	Breed[1]	Popn.[2] size	Gen.[3]	Sel.[4] trait	Basis of estimate
4 & 8 wk wt (Siegel, 1963b)	.94	WPR	S	4	8 wk wt	Response
6 & 9 wk wt (Merritt et al., 1966)	1.07	OMC	L	6	9 wk wt	Response
5-9 wk gn & 5 or 9 wk wt (Pym and Nicholls, 1979)	.50, .91	Synth	S	5	5-9 wk gn	Response

[1] OMC = Ottawa Meat Control; Synth = synthetic; WPR = White Plymouth Rock.
[2] Popn. size = population size; S = small; L = large.
[3] Gen. = generations of measurement.
[4] Sel. trait = selected trait.

Correlated responses in body weights or gain at ages other than the selected weight or gain have been reported as well. Divergent selection for 12-week weight of Silver Oklabar chickens for up to 15 generations gave rise to correlated responses in six-week body weight (Godfrey and Goodman, 1955; Maloney et al., 1963a, 1963b, 1967). There appeared to be better response in six-week weight of the line selected for high as opposed to low body weight (48 vs. 22 percent of 12-week weight). Similar selection of White Plymouth Rock chickens for eight-week weight for up to 27 generations caused correlated changes of similar magnitude for four-week weight of the high and low weight lines (5-6 g per generation) (Dunnington and Siegel, 1985). Ten generations of selection for five- to nine-week weight gain of a synthetic line of chickens improved weight gain 306 g or 38 percent (Pym et al., 1984). Correlated increases in five-week weight were 67 g (14 percent) and in nine-week weight were 374 g (29 percent). Differences of 120 g, 370 g, and 608 g in three- and six-week weights and three- to nine-week weight gain, respectively, arose between two lines of Bresse Pile chickens selected divergently through 20 generations for nine-week weight (Ricard and Leclercq, 1985). These responses give strong support to the high estimated and realized genetic correlations reported above.

Selection For Juvenile And Adult Growth In Opposite Directions

Efficiency of meat production and of reproduction of broiler chicks is enhanced by rapid juvenile growth in the broiler but minimal body size in the parent, especially the broiler mother. Achievement of this goal via genetic procedures requires modification of the growth curve. Three attempts to do this in meat strains of chickens have been reported.

White Wyandottes were selected through four generations for high eight-week and low 20-week weights (Gyles and Thomas, 1963). Details concerning this experiment are limited; however, efforts were reported to have been unsuccessful.

Lines originating from the Ottawa Meat Control strain were selected through 14 generations for high 63-day weight, high 147-day weight, or high 63- and low 147- day weights (Merritt, 1974). Comparison of the performances of the selected lines and the control line revealed that selection for high 63-day weight and low 147-day weight did increase juvenile weight but did not reduce mature weight. However, such selection did minimize the correlated increase in mature weight. The growth curve of this line was changed.

In another investigation, lines derived from the Bresse Pile strain were selected for one of the four combinations of high or low eight-week weight and high or low adult (36-week) weight for at least ten generations (Ricard, 1975). This study was successful in increasing one weight while reducing the second in the two lines which had weights selected in opposing directions. These two lines had growth curves altered.

In all cases it was obvious that positive genetic correlations hindered genetic change if the two weights were selected in opposite directions. Greater changes were observed in lines in which only one weight was selected or in which both weights were selected in the same direction.

Relationships Between Growth And Meat Production

Carcass and meat yield. Rapid growth was the major factor increasing yields of dressed and eviscerated carcasses representing 44 different strains and crosses (Jaap et al., 1950a, 1950b). For each unit increase in live weight, dressed and eviscerated carcass weights (giblets excluded and neck included) increased by .913 and .727 units, respectively. Small differences in carcass yields were found among various strains and crosses. In particular, Cornish or Cornish cross broilers had slightly superior carcass yields.

In studies of Bresse Pile broiler cockerels and Cornish broilers of both sexes, Ricard and Rouvier (1967, 1969) estimated full-sib genetic correlations between fasted body weight and weights of body parts. Correlations of live weight with weights of carcass parts were high (usually .9+); they were lower with abdominal fat and giblet weights (.2-.5). Principal components analyses revealed general factors, independent of one another, which accounted for genetic variation in live weight. The first, a major factor, was one of realized growth accounting for 65-83 percent of total variation in live weight; the second, a specific factor for fattening, accounted for an additional 9-12 percent of the variation. Other factors of lesser importance seemed related to dressing percentage and to measures of meatiness. Hence, as the chicken grows, the amounts of cuts of meat and of viscera increase; however, variations in proportions of meat cuts and of abdominal fat exist that are independent of growth rate.

Ricard and Leclercq (1985) compared yields of partly eviscerated carcasses of nine-week-old broilers from lines selected for high or low abdominal fatness (percentage of live weight) and for high or low body weight. The abdominal fat lines differed three-fold in abdominal fatness and had similar carcass yields (86.3 vs. 86.1 percent). The high and low weight selected lines differed in carcass yield (85.2 vs. 81.7 percent). The latter two lines of chickens differed greatly in live weight (1401 vs. 586 g). Unfortunately, carcass yield percentages at similar weights were not available, precluding the opportunity to determine whether the difference was due to differences in size or in rates of growth.

Only two estimates of genetic correlation between body weight and dressing percentage have been reported. Full-sib based estimates for Athens-Canadian randombred chickens were .55 for three nonselected generations (Muir, 1963) and .28 for five selected generations (Goodman, 1973). Unfortunately, there are no realized values for the genetic correlation between weight and dressing percentage to verify the above estimates.

Chemical components. Literature concerning the genetic relationship between growth rate and chemical composition of the carcass, either dressed or eviscerated, is sparse. In addition, comparisons are based on age, thus preventing comparisons at similar weight if differences in growth rate are also present. The increase in fatness as growth continues has been illustrated by Leeson and Summers (1980).

Genetic correlations between 23- to 44-day rate of gain and percentage chemical components of the carcasses of 529 female broilers, based on both sire and dam variance components, were estimated by Friars (1984). Rate of gain was negatively correlated with fat percent (-.39) and positively correlated with percentages of protein (.53), moisture (.32), and ash (.14).

Rapid and slow growing lines of White Plymouth Rock chickens fed *ad libitum* were slaughtered at around six weeks of age and proximate analyses of entire carcasses were determined (Proudman et al., 1970). Average body weights of the rapid and slow growth groups were 672 g and 345 g, respectively. Moisture, protein, fat and ash percentages were 68.1, 20.7, 6.9 and 3.0 for the rapid growth strain, and 69.8, 20.6, 4.8 and 3.1 for the slow growth strain. Unfortunately comparisons of the groups at similar body weights under *ad libitum* feeding were not available.

Chemical composition of entire carcasses was determined after five generations of selection for gain from five to nine weeks of age (Pym and Solvyns, 1979). A nonselected control strain was included for comparison. Percentages of moisture, protein, fat, and ash for the two lines were not significantly different when compared either at nine weeks of age or at similar weight around nine weeks of age. These lines differed in nine-week weight: 1565 vs. 1254 g in males and 1291 vs. 1067 g in females. Determinations of body lipid for these strains after nine generations of selection (Pym, 1985) revealed the rapid gain line to be slightly leaner than the control line throughout much of the growing phase.

The results of Proudman et al. (1970) suggest that rapid gain may give rise to increased fatness if the difference in size between the rapid and slow growing lines is disregarded. The results of research conducted by Pym (1985), in which size differences are considered, do not support observations of Proudman et al. (1970).

Abdominal fat. In excess, abdominal fat in broilers is detrimental to carcass quality and production efficiency. Not only the quantity but also the proportion of abdominal fat in the broiler increases with growth to ten weeks of age (Leeson and Summers, 1980). This feature interferes with determination of relationships between growth and abdominal fatness.

Genetic correlations between measures of abdominal fatness and growth have been estimated in several studies. These are listed in Table 25.9. Correlations involving abdominal fat weight are larger than those for abdominal fat proportion. This appears to be at least partly due to the part:whole relationship because reduction of the body weight by the weight of the abdominal fat reduced the correlation between body weight and abdominal fat weight slightly (Becker et al., 1984; Cahaner and Nitsan, 1985). With regard to correlations with abdominal fatness, weights at younger ages have higher correlations than weights at older ages, live weights have higher correlations than carcass weights, and weights have higher correlations than postbrooding weight gains. In fact, weight gains tend to be negatively correlated with abdominal fatness. Few of the correlations presented are greater than .5; however, they indicate that selection to increase body weight will give rise to increases in abdominal fatness.

Correlated responses in body weight due to divergent selection for abdominal fatness are shown in Table 25.10. Responses indicate that selection to reduce abdominal fatness does not inhibit response to selection for increased body weight unless there are reductions in

Table 25.9. Estimated genetic correlations (r_g) between measures of growth and abdominal fatness.

Traits (Reference)	r_g	Breed[1]	Popn.[2] size	Gen.[3]	Age	Basis of estimate[4]
Live & fat wt	.48	BrPi	S	1	11 wk	S+D
(Ricard and Rouvier, 1967)						
Live & fat wt	.20	C	S	1	59 d	S+D
(Ricard and Rouvier, 1969)						
Live & fat wt	.48	C	M	1	7 wk	?
Carcass & fat wt	.30					
(Becker et al., 1979)						
4-7 wk gain & fat %	-.30	Br	M	1	44 d	?
(Friars et al., 1983)						
Live & fat wt	.76, .40, .41	WR	M	1	50 d	S, D, S+D
Live less fat & fat wt	.72, .27, .42					
Carcass & fat wt	.68					
(Becker et al., 1984)						
Fat & 4, 6, 7 wk wt	.49, .30, .14	C, WR	M	1	4, 6, 7 wk	S
4-7 wk gain & fat wt %	-.07, -.24					
Carcass & fat wt %	.02, -.19					
Fat % & 4, 6, 7 wk wt	.33, .10, -.05					
(Chambers et al., 1984)						
Live & fat wt	.35	WR	S	1	9 wk	S+D
Live less fat & fat wt	.26					
(Cahaner and Nitsan, 1985)						
Live & fat wt	.45	C	M	1	46, 47 d	S
Live wt & fat %	.21					
(Leenstra et al., 1986)						
Live & fat wt (pens)[5]	.58, .54	C	M	1	42-44 d	S, D
Live & fat wt (cages)[5]	.55, .30					
Live wt & fat % (pens)	.36, .24					
Live wt & fat % (cages)	.47, .09					
(Leenstra and Pit, 1988)						

[1] BrPi = Bresse Pile; Br = broiler strain; C = Cornish; WR = White Rock.
[2] Popn. size = population size; S = small; M = medium.
[3] Gen. = generations of measurement.
[4] S = sire; D = dam; S+D = sire and dam.
[5] pens = tested in pens; cages = tested in cages.

either selection intensity or efficiency of selection procedures. These results suggest that realized correlations between these traits may be asymmetrical.

Correlated responses in abdominal fat deposition in four lines selected for body weight to modify their growth curves were examined by Ricard (1978). Change was induced by selection for high or low eight-week weight and either high or low 32-week weight for at

Table 25.10. Correlated responses in juvenile body weight associated with divergent selection for abdominal fatness or plasma VLDL[1].

Trait Selected (Reference)	Trait Correlated	Line response Fat	Line response Lean	Breed[2]	Popn.[3] size	Gen.[4]	Age (wks)
Fat		35.5 g	8.9 g	WPR	S	7	9
	Weight 9 wk	1.97 kg	1.93 kg				
(Leclercq, 1988)							
VLDL		.374	.060				
	Fat	36.6 g	14.9 g	C	S	7	7
	Weight 7 wk	2.24 kg	2.34 kg				
(Whitehead, 1988a)							
Fat		25.2 g	12.8 g	WR	S	5	8
	Weight 8 wk	2.02 kg	2.03 kg				
(Cahaner, 1988)							
Fat		29.9 g	15.9 g	C	S	3	7
	Weight 7 wk	1.87 kg	1.81 kg				
(Cahaner, 1988)							

[1] VLDL = very low density lipoprotein.
[2] C = Cornish; WPR = White Plymouth Rock; WR = White Rock.
[3] Popn. size = population size; S = small.
4 Gen. = generations of measurement.

least five generations. Analysis of the abdominal fatness of the one control and four selected lines revealed the following: the control line was at least as fat as any of the other lines; the line selected for low juvenile and high adult weights was the leanest; high juvenile weight appears to promote abdominal fat deposition; high adult weight appears to reduce abdominal fat deposition. Some of these observations are not readily apparent unless allowances are made for differences in body size.

Conformation and skeletal measures. The amount of meat derived from a chicken increases as it grows. Breeders have been interested in increasing the meat yield of the chicken and in improving the appearance of the carcass. Consequently, there has been interest in the genetic relationship of growth with conformation and skeletal dimensions. Frischknecht and Jull (1946) studied amounts of breast meat and live and dressed grades of purebred Cornish, New Hampshire, and Barred Plymouth Rock chickens and various crosses and backcrosses at 12 weeks of age. Faster growing chickens had higher carcass grades. In fact, the mean 12-week weight of the chickens in each grade tended to increase by 100-200 g for each increment in grade. There were similar increases not only in amounts but also in percentages of breast meat, cross-section breast area, shank length, keel length, body depth, and breast depth and width. Body dimensions would be expected to increase as the chicken grows; however, growth also favors greater carcass meatiness.

A principal components analysis using genetic correlations among traits of Cornish chickens at 59 days of age (Ricard and Rouvier, 1968) revealed the following: a major size component, best reflected by trunk length and correlated with live weight, that accounted for 56-60 percent of the variation in live weight; a carcass compactness component, best reflected by breast angle and correlated with shank width, that accounted for a further 18-20 percent of live weight variation; two oppositions, one between shank and thorax widths, and the other between chest and shank growth, accounting for proportions of the remaining variation.

Estimated genetic correlations between measures of growth and of conformation and skeletal dimensions have been reported in several studies. The relationship between body weight at ten weeks of age and breast angle was examined in 4807 purebred and crossbred broilers of New Hampshire and Silver Oklabar parents (Brunson et al., 1956). The estimated genetic correlations based on sire and dam components but excluding the effects of their interaction varied (.27-.79 for males; .47-1.05 for females). The authors concluded that selection for body weight should also produce a reasonable improvement in breast width. Other estimated genetic correlations between measures of growth (body weights) and of conformation and skeletal dimensions are tabulated in Table 25.11. Correlations between body weights and either breast width or breast angle are low, ranging from -.08 to .48. With the exception of one estimate between body weight and shank width (.39), estimates between body weight and skeletal dimensions were high (.5 or higher, often >.6).

Realized genetic correlations between body weight and breast angle and, in one study, keel length and shank length as well, are presented in Table 25.12. Realized correlations between body weight and breast angle tend to be higher (.22-.60) than estimated values from Table 25.11. However, the two realized values for correlations of body weight with keel length and shank length, both .50, tend to be lower than estimated values.

A few studies have provided estimates of correlated responses to selection involving combinations of growth and conformation or skeletal dimensions. Jull and Glazener (1946) selected Barred Plymouth Rock and New Hampshire ten-week-old chickens divergently for shank length. Progeny of the long- and short-shanked parents differed in four- and ten-week body weight indicating a positive relationship. Merritt et al. (1963) selected 63-day-old chickens of three lines derived from the Ottawa Meat Control population through four generations for breast angle. An increase in breast width was observed; however, no differences in body weight and reductions in keel and shank lengths were observed. Lines of White Plymouth Rock chickens were selected divergently for either breast angle or body weight at eight weeks of age for four generations (Siegel 1962b). The lines selected for breast angle differed by 6-7° for this trait and by 86-119 g in body weight. The body weight selected lines differed by 294-320 g in weight and 5-6° in breast angle. These results indicate that the realized correlations are symmetrical. After 27 generations of divergent selection for eight-week body weight, Dunnington and Siegel (1985) reported that the average change in breast angle per generation was .15-.17° for the high weight line and -1.05 to -1.13° for the low weight line. Twenty generations of divergent selection of Bresse Pile chickens for body weight (Ricard and Leclercq, 1985) caused high lines to exceed low lines by 644-813 g in body weight and 4.3-4.5° in breast angle at nine weeks of age.

In summary, body weight and skeletal dimensions, with the possible exception of shank thickness, are highly positively genetically correlated. Selection for increased growth or body weight will increase skeletal dimensions and meat yield. Conformation, as reflected by breast angle or width, is also positively genetically correlated with body weight; however, the correlation is small. Values tend to be similar whether the relationships between weight and breast angle are estimated or realized correlations.

Table 25.11. Genetic correlation estimates (r_g) between measures of growth and of conformation and skeletal dimensions.

Traits (Reference)	r_g	Breed[1]	Popn.[2] size	Age (wks)	Basis of estimate[3]
Wt & breast width	.10	NH	?	12	S+D
Wt & keel length	.79				
Wt & shank length	.87				
(Lerner et al., 1947)					
Wt & breast width	-.08	NH	?	12	S+D
Wt & keel length	.65				
Wt & shank length	.87				
(Abplanalp et al., 1960)					
Wt & breast width	small	NH	M	8, 12	?
Wt & keel length	.50				
(Dillard et al., 1953)					
Wt & breast angle	.24, .48, .39	NH, SO	S	9	S, D, S+D
(Godfrey and Goodman, 1956)					
Wt & breast angle	.21	OMC	L	9	S+D
(Merritt et al., 1963)					
Wt & breast angle	(S) .12, .07, -.08 (D) .26, .29, .14	OMC	L	6, 9, 21	S, D
Wt & keel length	(S) .67, .71, .70 (D) .73, .70, .51				
Wt & shank length	(S) .78, .80, .63 (D) .80, .82, .62				
(Merritt, 1968)					
Wt & breast angle	.24	C	S	59 d	S+D
Wt & keel length	.84				
Wt & shank length	.70				
Wt & trunk length	.84				
Wt & leg length	.66				
Wt & leg girth	.78				
Wt & shank width	.39				
Wt & thorax width	.80				
Wt & body depth	.82				
(Ricard and Rouvier, 1968)					
Wt & breast length WPR	.91	WPR, LS, x	M	8	S+D
LS	.80				
x1	.80				
x2	.86				
(Ayoub et al., 1980)					

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[1] C = Cornish; LS = Light Sussex; NH = New Hampshire; OMC = Ottawa Meat Control; SO = Silver Oklabar; x = crosses.

[2] Popn. size = population size; S = small; M = medium; L = large.

[3] S = sire; D = dam; S+D = sire and dam.

Relationships Between Growth And Feed Traits

Feed conversion can be defined as feed/gain and feed efficiency as the inverse, gain/feed. Due to inconsistencies in the use of these terms in the literature, discrepancies between terms as used here and in the literature cited may arise. Difficulty in measuring feed consumption or conversion on a weight constant basis necessitates the adoption of age constant measures. Concern about this practice has been expressed by Sorensen (1984) and Chambers and Lin (1988). In fact, Pym (1985) presented results in both forms. Where possible, measures of feed conversion or efficiency corrected for differences in weight or size will be noted below. Reports of genetic relationships between measures of growth and of feed consumption and conversion are abundant.

Genetic correlations have been estimated by several investigators using either sire or sire and dam components. Estimates of correlations between broiler weights and gains and feed consumption were generally high (.5-.9) (Pym and Nicholls, 1979; Chambers et al., 1984; Friars, 1984). Corresponding correlations between the growth measures and feed conversion were negative and lower (-.2 to -.8), with the low values associated with the initial test weight (Fox and Bohren, 1954; Pym and Nicholls, 1979; Friars, 1984). Values for feed efficiency were generally similar but positive due to the nature of the ratio (Chambers et al., 1984). The one exception was -.14 between initial test weight and feed efficiency. Generally, values in the literature are in good agreement, considering the differences among strains, ages of measurement, and management systems used.

Realized genetic correlations have been reported by Wilson (1969) and by Pym and Nicholls (1979) for gain and feed traits. Following one generation of selection for either

Table 25.12. Realized genetic correlations (r_g) between measures of growth and of conformation and skeletal dimensions.

Trait						
Selected (Reference)	Correlated	r_g	Breed[1]	Popn.[2] size	Gen.[3]	Age (wks)
Weight	Breast angle	.56	WPR	S	4	8
Breast angle	Weight	.45				
(Siegel, 1962b)						
Breast angle	Weight 4 wk	.22	WPR	S	4	4, 8
(Siegel, 1963a)						
Weight	Breast angle	.22	OMC	L	6	9
	Keel length	.50				
	Shank length	.50				
(Merritt et al., 1966)						
Weight	Breast angle	.60	WPR	S	4	8
Breast angle	Weight	.22				
(Carte and Siegel, 1968)						

[1] OMC = Ottawa Meat Control; WPR = White Plymouth Rock.

[2] Popn. size = population size; S = small; L = large.

[3] Gen. = generations of measurement.

gain or feed conversion from 5-10 weeks of age in Athens-Canadian randombred chickens, Wilson (1969) reported a value of -.51. Pym (1971, cited by Pym and Nicholls, 1979) obtained a similar value (-.52) in chickens 1-4 weeks of age, and stated that feed conversion could be improved twice as effectively by direct selection as by selection for weight gain. Wilson (1969) predicted that selection for gain is three times as effective for increasing gain as selection for feed conversion; however, selection for gain is 75 percent as effective as direct selection for improving feed conversion. If the discrepancy between studies is not due to error in estimates of the parameters, it will be difficult to resolve. Lines were selected for five generations for weight gain or feed consumption or feed conversion from 5-9 weeks of age (Pym and Nicholls 1979); the correlations obtained were .71 for gain and feed consumption, and -.40 for gain and feed conversion. In general, the realized and estimated correlations are consistent.

Correlated responses which provide further information regarding the relationship between growth and feed efficiency are available from selection experiments. Proudman et al. (1970) compared growth, feed efficiency, and efficiency of dietary energy and protein utilization of lines of White Plymouth Rock chickens selected for high and low eight-week weight. The high line was either fed *ad libitum* or restricted to either the same feed consumption or the same growth as the low line. The high line was almost twice as heavy when fed *ad libitum*, and more efficient whether fed *ad libitum* or restricted in feed intake. Guill and Washburn (1974) demonstrated that response to divergent selection for feed efficiency in two strains differed little when weight was allowed to vary or was maintained constant. This suggests that the genetic relationships between growth rate and feed efficiency were not strong. In strains selected for 12 generations, Pym (1985) demonstrated the following: selection for weight gain from 5-9 weeks of age reduced male and female feed conversion by .28 and .37 on an age basis, and by .69 and .77 on a weight basis; selection for feed consumption from 5-9 weeks of age increased male and female weights by 113 and 87 g at five weeks, and by 333 and 350 g at nine weeks, and increased feed conversion by .64 and .47 on an age basis and by .48 and .12 on a weight basis; selection for feed conversion from 5-9 weeks of age increased male and female weights by 55 and 48 g at five weeks, and by 279 and 244 g at nine weeks, and reduced feed conversion by .55 and .58 on an age constant basis and by .87 and .85 on a weight basis. Comparison of the lines revealed that selection for gain caused major increases in weight and increases in feed consumption, whereas selection for feed conversion increased weight with essentially no change in feed consumption. In conclusion, selection for weight or gain will increase feed efficiency; however, there is variation in efficiency that is not related genetically to growth rate.

Changes in metabolism related to either growth rate or to feed consumption or conversion have been shown to arise as a consequence of selection. In a review of many aspects of this topic, Siegel et al. (1984) reported the following: selection of meat-type chickens for weight has increased appetite such that it is near gut capacity with *ad libitum* feeding; increased appetite has the consequence of more meals per day, with evidence that satiety mechanisms are either absent or are functioning improperly; in addition to increased feed consumption, water:feed ratios of chicks immediately posthatch are increased; selection for faster growth with the associated greater obesity should interfere with optimum efficiency but the net result of changes related to improved growth give rise to improved efficiency. Correlated changes observed with faster growth that enhance efficiency include the following: more efficient use of energy and some amino acids by the embryo, reduced nutrient requirement for maintenance to market weight due to faster growth, reductions in oxygen requirement, increased rate of food passage, improved intestinal absorption of glucose, and improved body temperature regulation. Faster growing chickens prefer and may require diets with higher levels of protein.

Other research supports some of these principles. Meat-type chickens selected for faster growth utilize dietary energy and protein more efficiently (Proudman et al., 1970; Pym et al., 1984). Similar benefits were found when feed conversion was reduced by selection but not when feed consumption was increased by selection (Pym et al., 1984). Tomas et al. (1988) have shown that proportionate muscle protein breakdown is reduced by selection to increase weight gain or feed efficiency.

Stewart et al. (1980) demonstrated that ten generations of selection for low oxygen consumption caused chickens to grow faster. Provided dietary protein levels were adequate, these chickens were more efficient than their counterparts selected for high oxygen consumption.

Relationships Between Growth And Reproduction

Literature related to the genetic aspects of this topic have been reviewed in depth by Marks (1985) and by Siegel and Dunnington (1985). Little additional information has appeared since their reports.

Male reproduction. Genetically increased growth rate or juvenile body weight has negative influences on reproductive traits of the cockerel. Estimated and realized genetic correlations between body weight and motility of spermatozoa are negative. Ejaculates from cockerels of lines selected for high as opposed to low juvenile weight tend to contain spermatozoa with lower metabolic rates and higher percentages of dead and abnormal members. Males from high weight lines produce ejaculates of greater volume but lower concentrations of spermatozoa. Males from high weight lines have exhibited reduced libido and mating frequency; however, no correlated responses in body weight were found to accompany divergent selection for mating frequency.

Female reproduction. The net effects of genetic increases in growth rate or juvenile body weight on female reproduction are negative. Some of these effects appear to be positive; however, it appears that physiological imbalances nullify any beneficial influences. From endocrine studies, it is evident that body weight is positively correlated with pituitary size and target organ sensitivity. A positive relationship is apparent between body weight and ova production. There is a positive genetic correlation between egg weight and body weight; Kinney (1969) reported correlations of .26 with eight-week weight and .33 with adult weight. Estimated genetic correlations and responses to selection indicate that in meat chickens a negative relationship exists between juvenile body weight and normal egg production. The apparent discrepancy between increased ova production and reduced egg production is accounted for by increased incidence of abnormal eggs (double yolks, extra-calcified shells, compress-sided eggs collectively called EODES or erratic oviposition and defective egg syndrome), internal laying, and progressive regression of developing follicles. Further support of the latter point has been presented by Hocking et al. (1987). Sexual maturity, the age at onset of egg production, is influenced by age, body weight, and body composition. Correlated responses in age at sexual maturity to selection for juvenile body weight vary, ranging from moderately positive to moderately negative. In long-term selection experiments, onset of maturity in low weight line pullets may even be inhibited. These chickens fail to attain the necessary body weight or composition.

Chromosomal aberrations. These aberrations, which result in gametes with an imbalance of chromosomal material, give rise to lethality, semisterility and intersexuality. These conditions tend to be self-eliminating but this feature also reduces fitness, especially hatchability. The following is known regarding chickens: heteropoloidy is about 7-8 times more frequent in meat-type than in other types of chickens, chromosomal aberrations have a higher incidence (two- to three-fold) in lines selected for high juvenile weight than in nonselected or low weight selected lines, and some types of aberrations (haploidy, triploidy) tend to be either maternal in origin or under maternal influence. Hormonal imbalances in the female may contribute to the origin of these defects.

In summary, selection for rapid growth tends to give rise to negative effects on male and female reproduction and to enhance chromosomal aberrations which reduce hatchability. These effects are definitely negative; however, if parental populations are sufficiently large and optimal management is practiced, these influences should not preclude improvement of growth rate by genetic methods.

MEAT PRODUCTION

The meat or edible tissue from the chicken carcass consists primarily of muscle tissue. Giblets consisting of the neck and of organs such as the heart, liver, and gizzard are frequently included. Lipids of intracellular and intermuscular origin are included in meat, whereas lipids of subcutaneous and abdominal origin and from the skin may also be included. Some additional nutrients may be derived from the skeleton during cooking or if broth is obtained for making soup.

Meat production can be evaluated in terms of either yield or quality. Meat yield may be expressed as carcass yield (or dressing percentage), yield of specific carcass parts, and yield of specific tissues such as lean, fat, skin, and bone. Meat quality can be assessed by several methods: by carcass conformation or grade, sensory evaluation or flavor, juiciness, and tenderness of the cooked carcass or carcass part, and by chemical composition of the carcass to determine the amounts or proportions of protein, fat, moisture, and ash.

Measurement of meat yield. Carcass yield or dressing percentage is expressed as weight of the carcass as a percentage of the weight of the live chicken (usually fasted). This measure reflects the losses due to removal of feathers, blood, and offal.

Similarly, yield of carcass parts is expressed as the weight of the specific part — breast, back, wings, legs (thighs included), and giblets — as a percentage of carcass weight. These traits assist in evaluation of the proportions of high versus low value cuts.

Yield of specific tissues is determined by dissection of the tissue — muscle, fat, skin, and bone — and expressing the tissue weight as a percentage of the carcass. These measures aid in evaluation of the meatiness of the carcass.

Yield of carcass after cooking is expressed as a percentage of the carcass prior to cooking. The losses are due to evaporation of moisture, and to rendering and loss of adipose tissue during cooking.

Measurement of meat quality. Esthetic value of the carcass is appraised subjectively based upon fleshing, finish, conformation, and defects such as blisters and cysts, skin lesions, injuries including broken limbs and bruises, and crooked or deformed keels. Defects lead to downgrading and devaluation of the entire carcass; however, if subjected to further processing, much of the carcass can be marketed at top value.

Sensory properties of the cooked carcass or carcass part are evaluated by a trained panel of judges. Judges test samples of the meat for flavor, juiciness and tenderness.

Chemical composition of the carcass is analysed by proximate analysis to determine the proportions of moisture, protein, lipid, and ash. This measure leads to the destruction of at least part of the carcass; however, these measures are major determinants of the nutritional value of carcasses.

Carcass And Meat Yield

Breed and strain differences. Genetic differences exist among strains and lines of chickens for yields of dressed and eviscerated carcasses, of carcass parts such as breast, back, wings, and legs (drumstick and thigh), of edible meat (from deboned carcass or parts), and of meat, skin, and bone. The positive correlations between growth rate (or size) and yield reported by Jaap et al. (1950b), Davis and Hutto (1953), and Bouwkamp et al. (1973) tend to interfere with the accurate evaluation of such differences for either individual chickens or groups of chickens that differ in size. Such might not occur if it were more practical to slaughter single chickens or groups of chickens at the same live or carcass weight.

In studies of various breeds and their crosses, the Cornish breed or Cornish crosses have been superior with regard to yields of dressed and/or eviscerated carcass, breast meat, and edible meat (Jaap, 1941; Stotts and Darrow, 1952; Davis and Hutto, 1953; Hathaway et al., 1953; Morrison et al., 1954; Orr, 1955). Some comparisons included allowances for differences in body size. In one study (Moran et al., 1970) of broilers of Cornish and White Rock breeds and their crosses, only carcass grade was superior for Cornish x White Rock broilers. No group differences were apparent for yields of dressed and eviscerated carcasses, carcass parts, or for meat, skin, and bone. Comparisons among other breeds in the above studies plus those of Jaap et al. (1950b) and Essary et al. (1951) were less consistent; however, White Rock purebreds or crosses were superior to breeds other than Cornish.

Commercial broiler stocks and, in some instances, other breeds or stocks have also been compared with regard to yields of carcass and carcass parts. Stock differences were reported by Gyles et al. (1954), Bouwkamp et al. (1973), Merkley et al. (1980), Orr et al. (1984), Wabeck et al. (1984), and Gyles (1987). In a comparison of four crossbreds, Moran and Orr (1970) reported no differences when only first grade carcasses were included. Differences in yield of carcass parts reported by Bouwkamp et al. (1973) disappeared when comparisons were restricted to carcasses of similar size.

Early reports favored the Cornish breed and in some instances the Plymouth Rock breed. Frequently the New Hampshire breed exhibited lower meat yield. These results seem to account for the use of Cornish sire and White Rock dam lines for broiler production. The trend for commercial breeders to follow this practice may account for the small differences observed more recently among commercial stocks. The differences that are observed probably reflect differences in line development and improvement practices.

Heritability of carcass and meat yield. In this section, estimated heritabilities will be based upon sire and dam variance components unless indicated otherwise. Studies which report heritabilities for these traits are scarce; however, there seems to be consistency among the various estimates in spite of differences in strains, environments, and management.

Athens-Canadian randombred chickens in a diallel mating design were measured for eviscerated carcass yield through three generations prior to selection for this trait (Muir and

Goodman, 1964). The heritability estimate was .14 (nonadditive effects excluded); the normal estimate from a nested mating design was calculated to be .32. During five generations of selection for carcass yield, data were collected at ten weeks of age from 958 progeny of 180 dams and 70 sires (Goodman, 1973). The heritability estimate was .29. The response to three generations of selection was an increase of .62 percent in carcass yield (Muir and Goodman, 1964).

Three hundred and fifteen Bresse Pile cockerels were slaughtered, processed and sectioned into parts (Ricard and Rouvier, 1967). Heritabilities of weights of carcass parts corrected for differences in live weight were dressing loss .33, evisceration loss .25, breast .50, legs .31, wings .52, back and neck .30, shanks and feet .50, and head .31.

Three hundred Cornish broilers, 150 of each sex, were also studied by Ricard and Rouvier (1969). Heritabilities calculated for weights of carcass parts as ratios of live weight were dressing loss .16, evisceration loss .29 (intestines and mesentery) and .14 (other viscera), breast .53, legs .72 (thigh) and .67 (drumstick), wing parts .36 (arm) and .41 (forearm), shanks and feet .87, head .33, and giblets .60.

Broilers, 1356 cockerels produced by 74 sires and 305 dams, were slaughtered at 80 days of age and processed (Ricard, 1974). Heritability estimates for yields were eviscerated carcass .45, giblets .49, breast muscle .62, and leg muscle .58. For yields of leg, heritability estimates were muscle .46, skin .52, bone .60, and leg meat to bone ratio .54.

Three hundred commercial White Rock broilers slaughtered at two ages provided yield data for heritability estimation (Cahaner and Nitsan, 1985). Estimates of heritability for traits corrected for differences in live weight were evisceration loss .54, breast .45, thigh .31, drumsticks .49, wings .44, back and neck .46, head .38, shanks and feet .67, liver .50, gizzard .56, and carcass skin .37.

Eviscerated carcass and yield of leg and breast meat were measured in broilers from two strains selected divergently through three generations for abdominal fatness (Ricard et al., 1982). The high fat strain had twice as much abdominal fat as the lean strain. Correlated responses led to 1-2 percent lower eviscerated carcass yield and 2-3 percent lower breast meat yield in the fat compared to the lean strain.

In general, heritabilities of dressed and eviscerated carcass yields and losses were low (.1-.3). Estimates for the other measures were high (carcass part yields .3-.7, giblets .3-.9, meat, skin and bone yields .4-.6). It appears that the small amounts of variation in these traits and the need to slaughter the chicken to measure the trait are main factors limiting major genetic change.

Relationships of carcass and meat yield. Several studies have concerned the relationship between carcass and meat yield as well as other conformation and meat-related traits. Breast meat yield was studied by Blow and Glazener (1952) using New Hampshire chickens and by Frischknecht and Jull (1946) using Cornish, New Hampshire, and Barred Plymouth Rock chickens and their crosses. Body weight was the best single trait for predicting amount of breast meat. Breast width and area of a cross-section of the breast muscle were also of value for this purpose. Breast muscle cross-section area was the best criterion of market grade. Cornish crosses graded higher and had better yields of breast meat than non-Cornish groups. Jaap (1941) examined conformation measures and yield of edible meat from 1024 chickens of White Plymouth Rock and White Leghorn strains, of crosses of these strains, and of female crosses of these strains mated to Dark Cornish sires. Cornish-cross broilers had 1.5-1.9 percent higher edible meat yield. The best yielding groups had smaller body depths and keel and shank lengths relative to live weight. In the study of chickens of New Hampshire and Barred Plymouth Rock strains and their crosses by

Essary et al. (1951), Barred Plymouth Rocks had the highest breast yield and lowest wing and leg yields. The New Hampshire x Barred Plymouth Rock cross had the highest dressing percent (89.2); New Hampshire chickens had the lowest (86.3). Davis and Hutto (1953) noted that within groups of broilers from two synthetic strains and their crosses, there was a positive relation between breast yield and breast angle measurement. There was also a positive relation between live weight and yield of eviscerated carcass. There was no relationship between leg and breast yields. In the study of Ricard and Rouvier (1967), genetic correlations of live weight and weights of the carcass and its parts were positive and large (.74 or higher), with the exception of abdominal fat (.33 - .52). Correlations among weights of carcass and parts corrected for differences in live weights were much lower (<.57) and frequently negative. Legs had low correlations with other parts except wings (.40). Wings were positively correlated with breast (.32), but negatively correlated with back (-.46). Back was negatively correlated with breast (-.27). Viscera had a positive correlation with back (.24), but negative values with other parts (-.37 to -.56). Similar coefficients were reported among weights of carcass components by Ricard and Rouvier (1969), except values involving abdominal fat were lower and, in some instances, negative. Goodman (1973) reported a genetic correlation of .28 between live weight and dressing percentage (eviscerated carcass, no giblets).

To summarize, correlations between live weight and all carcass measures except abdominal fat are high and positive. Correlations among the weights of the parts are high and positive, again excluding abdominal fat. Among yields of the carcass and of its parts corrected for live weight, correlations are lower and frequently negative.

Chemical Components Of The Carcass

Breed and strain differences. Reports of strain differences are more numerous than heritability estimates for chemical components of the carcass. Twelve strains of broilers in the Arkansas Random Sample meat test were sampled and meat and skin of half carcasses were ground, blended, and analyzed for fat and protein (Goodwin et al., 1969). Carcasses of the strains differed in moisture, protein, and fat content. Composition of carcasses of broilers of Cornish and White Rock strains and their crosses was determined by Moran et al. (1970). Carcasses of White Rock broilers had 2-4 percent more fat on a dry matter basis than other stocks, with corresponding reductions of protein and moisture to accommodate this increase. Edwards and Denman (1975) found differences in quantities of moisture, protein, lipid, and ash among Light Brahma, White Plymouth Rock, White Leghorn, Jersey Black Giant, and Dark Cornish chickens. Light Brahmas grew slowest but were fattest. Dark Cornish were leanest. In a second experiment Dark Cornish and White Plymouth Rock strains were compared and again the Cornish breed was leaner. Carcass fatness of five commercial broiler stocks was measured by Becker et al. (1981). No differences were detected when ten broilers per sex of each stock were examined.

Heritabilities of chemical components of the carcass. Friars (1984) estimated genetic parameters of carcass composition of 529 female broilers from 196 dams and 31 sires. Heritabilities and genetic correlations based on sire and dam variance and covariance components were estimated. Heritabilities were moisture .38, protein .47, fat .48, ash .21. Genetic correlations were 1.03 for moisture with protein, -1.02 for moisture with fat, .52 for moisture with ash, -1.01 for protein with fat, .81 for protein with ash, and -.85 for fat with ash.

To summarize, some strains exhibit definite differences in carcass components. With the exception of ash, carcass components appear to be moderately to highly heritable. Fat is negatively correlated with the other components while remaining combinations are positively correlated. Correlations among carcass components are high.

Relationships involving chemical components of the carcass. Genetic correlations between chemical components of the carcass and weight gain, feed intake, and feed conversion were estimated by Friars (1984). Respective values for moisture, protein, fat and ash were .32, .53, -.39, and .14 with gain; they were -.18, -.06, .10, and -.17 with feed intake; they were -.63, -.80, .65 and -.40 with feed conversion. The three performance traits were measured between 23 and 44 days of age.

Correlated responses to selection for other traits have been observed for chemical components of the carcass. Fast and slow growing lines of White Plymouth Rock chickens fed *ad libitum* differed in carcass moisture, fat, and ash (Proudman et al., 1970). Lines of Athens-Canadian randombred chickens selected to differ in yolk cholesterol did not differ in carcass components (Washburn et al., 1976). Divergent selection for abdominal fatness for seven generations not only altered the selected trait but it also changed lipid (4.9-8.8 percent) and protein (.5-2.0 percent) content of male carcasses (Leclercq, 1983). Five generations of selection for feed consumption, weight gain, or feed conversion from 5-9 weeks of age was shown to increase fatness of the feed consumption line and to reduce fatness in the feed conversion line (Pym, 1985). In spite of continued selection, there was little change in chemical components of the carcasses in later generations. Divergent selection of a commercial broiler strain through four generations for plasma very low density lipoprotein also altered lipid (5.4-5.6 percent) and protein (1.0-1.1 percent) of the total body (Whitehead and Griffen, 1986).

Carcass components appear to be more closely related to feed efficiency than to gain or feed intake. Components can be changed through selection for other traits such as plasma very low density lipoprotein, feed consumption, feed conversion, and abdominal fatness.

Sensory And Cooking Qualities

Information regarding genetic aspects of these traits in chicken meat is sparse. No genetic information could be found for sensory or organoleptic traits. Heritability estimates based on sire and dam variance components for weight losses were estimated as thaw loss .44, volatile cooking loss .27, and drip loss .33 (Friars, 1984). Heritabilities for percentage losses were volatile loss -.22 and -.24, drip loss .29 and .02, and combined volatile and drip losses .36 and .25 for male and female broilers, respectively (Marini and Gyles, 1973). Friars (1984) also presented genetic correlations with other traits for weight of thaw loss; these were .80 with fat-free carcass weight, -.36 with carcass fat, .68 with volatile cooking loss, and -.21 with drip loss. Corresponding values for volatile cooking loss weight were .97 with fat-free carcass weight, -.45 with carcass fat, and -.28 with drip loss. Values for drip loss were -.26 with fat-free carcass weight, and .87 with carcass fat. Thaw and volatile losses are positively related to carcass moisture; drip losses are similarly related to carcass fatness.

Conformation And Skeletal Measurements

The use of live body measures as indicators of conformation was advocated by Jaap and Penquite (1938). In flocks of six different breeds of chickens, differences among families were detected for the ratio between shank length and body weight, and breed differences were observed for this ratio and for body depth (Jaap and Thompson, 1940). Wise (1970) noted that broilers had similar proportions by weight of the various body parts as layers when compared at the same weight; however, broilers had shorter bones and more compact body parts. Jaap and Thompson (1940) also reported keel length differences to be inherited and that selection for various traits improved body shape. Collins et al. (1950) observed a high positive correlation between breast angle and live grade, a positive correlation between breast width and body weight, and a higher proportion of broad-breasted progeny from broad-breasted than from narrow-breasted parents. Chickens from small eggs had poorer meat conformation, with a higher ratio between shank length and body weight, than those hatched from large eggs (Wiley, 1950). However, selection for egg weight did not alter the ratio between shank length and body weight of subsequent progeny.

Many of the body measurements that have been studied are good indicators of skeletal size. These include keel length and breast length, trunk length, shank length and leg length, leg circumference, body depth, and thorax width. Common properties that warrant grouping these traits include high heritabilities and genetic correlations with body weight and with each other. Average heritabilities for these traits are within the range of .4-.6, and are usually about .5 (Lerner et al., 1947; Dillard et al., 1953; Kan et al., 1958; Abplanalp et al., 1960; Merritt, 1966; Merritt et al., 1966; Ricard and Rouvier, 1968; Ayoub et al., 1980). Some estimates based on sire and dam variance components were similar to those obtained by regression of offspring on parent (Kan et al., 1958; Merritt, 1966). The genetic correlations of these traits were generally high (.6 and higher), not only with body weight but also with each other (Lerner et al., 1947; Dillard et al., 1953; Kan et al., 1958; Abplanalp et al., 1960; Merritt, 1966, 1968; Merritt et al., 1966; Ricard and Rouvier, 1968; Ayoub et al., 1980). Their genetic correlations with shank thickness were lower (often .4 or less), and with breast width they were even lower and in some instances even negative (Lerner et al., 1947; Abplanalp et al., 1960; Merritt, 1966, 1968; Merritt et al., 1963, 1966; Ricard and Rouvier, 1968). Genetic correlations of keel length and shank length with egg production traits were small (less than .3) and sometimes negative (Merritt, 1968).

Breast width, frequently used to refer to breast angle, is not always measured using the breast angle metre. Collins et al. (1950) found that breast angle was highly correlated (.8+) with breast width measured at 3.0 cm above the keel of the standing chicken. Moreover, the two measures had similar correlations with live grade. However, thorax width appears to be biologically different from breast width. Phenotypic correlations between thorax width and breast angle were low (.20 for males, .29 for females) and these traits differed in their correlations with other traits (Ricard and Rouvier, 1968).

Heritabilities of breast width estimated from sire and dam variance components averaged .38 (Lerner et al., 1947; Dillard et al., 1953; Brunson et al., 1956; Godfrey and Goodman, 1956; Siegel and Essary, 1959; Abplanalp et al., 1960; Siegel, 1962c; Merritt, 1966; Ricard and Rouvier, 1968). Estimates derived from sire variances were .30 and .25 at ten weeks and six months of age, respectively (Cook et al., 1956). Heritabilities estimated from regression of offspring on parent averaged .30 (Dillard et al., 1953; Godfrey and Goodman, 1956; Siegel, 1962c; Merritt, 1966). Realized heritabilities have been reported as .24 for males and .21 for females by Siegel (1962b), and as .31 by Siegel (1962c). Carte and Siegel (1968) reported correlated realized heritabilities of .40 for males and .35 for females in lines selected divergently for eight-week weight.

The genetic relationships of breast width with other traits have been investigated. The intensity of investigation varies depending upon the related trait. Genetic correlation estimates are positive but generally low between body weight and breast width. Dillard et al. (1953) reported a slight correlation. Estimates reported by Lerner et al. (1947), Brunson et al. (1956), Godfrey and Goodman (1956), Siegel and Essary (1959), Abplanalp et al. (1960), Merritt et al. (1963, 1966), Siegel (1963a), and Merritt (1968) ranged from -.08 to .82 and averaged .21. Realized correlations between breast angle and body weight were less numerous ranging from .21 to .68 and averaging .42 (Siegel, 1962a; Merritt et al., 1966; Carte and Siegel, 1968). In a double divergent selection experiment for either body weight or breast angle at eight weeks of age (Siegel, 1962b), angles differed by 6-7° in the lines selected for breast angle and by 5-6° in the lines selected for body weight. Four generations of selection for breast width gave no change in body weight, but correlated reductions in keel and shank lengths were observed (Merritt et al., 1963). Siegel (1963a) reported values for breast width with semen volume (.15), semen concentration (.86), sperm motility (.35), age at sexual maturity in pullets (-.04), egg production (-.02), egg weight (.11), Haugh units (.0), and specific gravity of eggs (-.32). Merritt (1968) also reported values for breast width with egg production traits: egg production (.18), survivor egg production (.01), percent egg production (.06), percent survivor egg production (-.02), age at first egg (-.37), and egg weight (.0). Direct selection for breast width was compared with progress from selection for an index which included breast width, body weight, keel length and shank length (Abplanalp et al., 1960). Response of breast width to selection was 20 percent greater for the index than for breast width alone. Direct selection caused correlated reductions in egg production and hatchability and delayed sexual maturity.

For shank thickness, Ricard and Rouvier (1968) reported a high heritability (.90), but low genetic correlations (.4) with other traits except leg circumference (.50).

The research reported above and the principal components analyses by Ricard and Rouvier (1968) indicate that if body weight is available as an indicator of growth, there is usually little additional information to be gained from skeletal measurements. If carcass conformation or meatiness is being evaluated, then breast width information will be a definite asset. Likewise shank thickness may be helpful for evaluating carcass compactness.

Carcass Defects

There are various defects that detract from the value of the carcass. Three of these, crooked keels, breast blisters and deep pectoral myopathy, are influenced by genetic as well as management factors. Descriptions have been provided for crooked keels by Warren (1937), breast blisters or keel bursae by O'Neil (1943), and deep pectoral myopathy or green muscle disease by Siller (1984).

Chickens tend to develop crooked keels before maturity (Warren, 1937). Crooked keels have lower bone ash than straight keels; however, no differences in ash of leg bones could be found between crooked- and straight-keeled chickens. Age of commencement of roosting, sharpness of perches, and degree of use of perches influence the expression of the heritable differences in tendency to develop crooked keels. The expression of inherited differences in straightness of keel is determined largely by the early roosting conditions. Inherited differences are evident from strain differences, differences among progenies of straight- and crooked-keeled parents, and differences in incidence created by divergent selection. There was no influence of keel condition on growth rate or fecundity. This defect is less common with current management practices, with no roosts being used for growing chickens and few, if any, provided for adults.

The so-called breast blister is a bursa that develops between the keel bone and the skin of the breast (O'Neil, 1943) in response to mechanical irritation or pressure. The contents of the bursa undergo degenerative changes and the serous fluid is replaced by fibrous tissue. A strong relationship between incidence and severity of keel bursae and body depth was demonstrated by Bird (1944). The pressure of the roosting chicken against the perch is highly correlated with the chicken's relative body depth. It was concluded that freedom from bursae in rapidly growing chickens can only be expected in families with inherited shallowness of body. Kondra and Cavers (1947) demonstrated the benefits of feather covering of the breast in reducing the incidence of keel bursae. Genetic improvement of feathering rate together with reduction of breast depth in chickens appears to be the most practical means of reducing breast blisters.

Deep pectoral myopathy, or degenerative myopathy of the *musculus supracoracoideus* (DMS), is an ischemic necrosis of the supracoracoid or deep pectoral muscle that is more common in meat-type than egg-type chickens (Siller, 1984). The genetics of this defect is discussed in Chapter 33. It appears that with the emphasis given to breast angle and body weights in breeding programs, DMS will become more common. When handling meat-type chickens, all efforts should be taken to avoid wing flapping in order to prevent the development of this defect.

Abdominal Fatness

Excessive abdominal fatness has been recognized as a major problem confronting the chicken meat industry. It represents a large waste in that it not only reduces carcass yield and feed efficiency, but it also leads to consumer rejection of chicken meat and adds to processing difficulties. Genetic aspects of abdominal fatness in meat-type chickens have been reviewed by Siegel (1984) and by Marks (1988). Comprehensive discussions of research for genetic reduction of abdominal fatness have been presented by Cahaner (1988), Leclercq (1988), Leenstra (1988), and Whitehead (1988a). These reviews are the basis for much of the information that follows.

Breed and strain differences. Breeds, strains within breeds, and commercial broiler stocks frequently differ in abdominal fatness. Unfortunately the differences demonstrated frequently involve strains that differ in weight at slaughter. These differences do not represent the differences that would have been observed if strains had been slaughtered at similar body weights.

Heritability of abdominal fatness. Heritabilities, either estimated or realized, are high (.5-.8). They tend to be similar whether for abdominal fat weight or percentage of carcass or live weight. There is little evidence of differences in heritability between either experimental and commercial strains, or paternal and maternal strains of broiler parents.

Response to divergent selection for abdominal fatness has been dramatic. A four-fold difference was found after seven generations of selection by Leclercq (1988). Two-fold differences in Cornish and in White Rock strains were found after three generations of selection by Cahaner (1988).

Relationships between abdominal fatness and other traits. Abdominal fatness and body weight are positively correlated, both phenotypically and genetically; however, the values of genetic correlations are not high (about .3). In fact, there has been little evidence of differences in body weight response to divergent selection for abdominal fatness or traits related to abdominal fatness.

Abdominal fatness and feed efficiency are negatively related both phenotypically and genetically. Selection to reduce abdominal fatness causes a correlated improvement in feed efficiency, and conversely, selection to improve feed efficiency causes a correlated reduction in abdominal fatness. Estimates of genetic correlations of feed consumption and efficiency with measures of abdominal fatness were reported by Chambers et al. (1984). Feed consumption from 4-7 weeks of age was positively correlated with abdominal fat weight (.42) and percentage of carcass weight (.24). Feed efficiency for the same period was negatively correlated with these measures of abdominal fatness (-.62 and -.69, respectively). Leenstra et al. (1986) reported estimated correlations between abdominal fat (corrected for differences in body size) and feed conversion from 21-42 days of age to be .19 for sire family means and .29 for dam family means. Similar genetic correlations based on sire components were estimated by Leenstra and Pit (1988) using data from four selected lines during four generations of selection. Values for feed conversion ratio with abdominal fat weight and percentage of live weight were .43 and .44, respectively. Compared to a line selected through four generations for weight gain from 21-42 days of age, lines similarly selected for either low abdominal fatness or low feed conversion ratio from 21-42 days of age had 1.45 and 1.00 percent less abdominal fat, .11 and .17 lower feed conversion ratios, and .50 and .70 higher dressing percentages, respectively (Leenstra and Pit, 1987). Whitehead (1988b) has demonstrated that lean and fat lines of chickens developed by divergent selection for plasma very low density lipoprotein levels differ in their utilization of nutrients for growth of fat vs. lean tissue.

Genetic correlations between abdominal fat and other lipid depots or total carcass lipid tend to be very high and positive (.6-.9). Correlated reductions in other lipid depots have been demonstrated to accompany selection to reduce abdominal fat.

Relationships between abdominal fat and measures such as carcass specific gravity, water space of the live chicken, thickness of the abdominal wall with adhering leaf fat, triglyceride and very low density lipoprotein content of plasma, and lipoprotein lipase of abdominal fat are positive but moderate to low in magnitude. Unless values can be determined for the live chicken, their use in selection programs is limited. Abdominal wall thickness and plasma very low density lipoprotein levels are positively genetically correlated with abdominal fatness as demonstrated by response to selection. So far, these traits appear to be the best predictors of abdominal fatness in the live chicken.

Selection to alter the growth curve of the chicken alters abdominal fatness levels (Ricard, 1978). Abdominal fat levels even to 16 weeks of age were lowest in a line selected for low juvenile and high adult weight.

Research has demonstrated the need for a minimum level of fatness, as reflected by abdominal fatness, for onset of sexual maturity. Genetic correlations between abdominal fatness and reproductive traits are generally not available; however, if the relationship involves a threshold and not a continuum, these correlations may be of little value. Genetic selection to change abdominal fatness can alter the fitness of the selected lines. After divergent selection for abdominal fatness the number of eggs produced did not differ (Leclercq, 1988). Similar selection for plasma very low density lipoprotein levels had similar effects, provided hens had been fed on a restricted basis during rearing (Whitehead, 1988a); however, feeding hens on an *ad libitum* basis gave rise to better egg production by hens of the lean line. In the French study (Leclercq, 1988), fat line hens produced larger eggs. This was contradicted by the results of Whitehead (1988a) showing that lean line hens produced heavier eggs, whether feeding during the rearing period was on an *ad libitum* or a restricted basis. Eggs from lean line hens have lower hatchability (Leenstra, 1988). This was demonstrated by Whitehead (1988a) when hens had been fed *ad libitum* during rearing. However, no hatchability differences were observed by Leclercq (1988) or by Whitehead (1988a) when hens had been fed on a restricted basis during rearing. Leenstra (1988) noted that low fat line hens produced fewer defective eggs (double-yolked, soft-shelled, extracalcified) than hens from lines selected for high body weight or low feed conversion ratio.

OTHER TRAITS RELATED TO GROWTH AND MEAT PRODUCTION

Feed consumption and efficiency. In the introduction of this chapter, it was stated that rapidly growing chickens require adequate feed to express their rapid growth potential. This situation also applies for the expression of optimum feed efficiency. In most instances, feed costs incurred for the production of meat-type chickens equal or surpass all other costs. Hence, any factor improving feed efficiency that can be economically applied will enhance financial returns to the broiler producer. Genetic improvement of feed efficiency is no exception.

The reader is referred to Chapter 34 for greater detail regarding physiological, metabolic, and other factors influencing feed consumption and efficiency. The material relevant to broiler production may be very briefly summarized as follows. Correction of age-constant feed efficiency measures for differences in body weight improves accuracy of evaluation of this trait. Feed efficiency heritability estimates are usually in the range of .4-.5; realized heritabilities are about .25. Much of the variability in feed efficiency can be explained by variation in growth rate but other factors are involved as well. Improved feed efficiency is associated with reduced abdominal and carcass fatness. Improvements in feed efficiency arise from reductions in feed wastage, increases in feed intake providing more nutrients to be used for growth, increases in digestibility or metabolizability of nutrients, reductions in maintenance energy requirements, increases in the availability of metabolizable energy for gain, reductions in energy content in carcass tissue by reducing fat content, and reductions in muscle protein breakdown enhancing the efficiency of protein accretion. The heritability of feed efficiency and its generally favorable relationships with other important broiler production traits encourages selection for this trait.

Effect of nutrition on the genetic variation of growth. Nutrition is not only important for optimum growth but also important for optimum expression of genetic variation of growth. Genetic variation in the nutrient requirements of the chicken has been reviewed by Nesheim (1975). The influence of diet on the response to selection for growth and efficiency has been discussed by Sorensen (1985). He concluded that there is genetic variation in dietary protein required for growth, there is little evidence of genetic variation in dietary energy requirements for growth, and that the realized heritability of growth rate tends to be lower if low nutrient density diets are fed.

Part of the basis for the first conclusion is the results of two generations of selection for eight-week weight in Athens-Canadian randombred chickens fed 14, 18 or 22 percent crude protein diets (Marks and Britton, 1978). Each four percent increase in dietary protein increased not only realized heritabilities by .2, but also increased responses to selection by

50-100 percent. In addition, Leclercq (1983) has demonstrated strain differences in such responses. Testing fat and lean lines developed by selection, and using one of four diets differing in protein content (15.2, 17.2, 19.1, and 21.1 percent) from 3-8 weeks of age, revealed reductions in growth rate of the lean line when fed the two lower protein diets. These diets did not differ in their effect on growth rate of the fat line. In contrast, Mahmoud et al. (1965) reported heritabilities based on male New Hampshire data that were lower when a higher (24 vs. 18 percent) protein diet was fed. Johnson et al. (1966) selected Athens-Canadian randombred chickens for eight-week weight for six generations in two lines. One line was fed a ration with 21.5 percent protein, the other a ration with 16.5 percent protein for testing progeny. When the two lines were tested on the same ration, the line selected for performance on the low protein ration grew faster but was also fatter.

Sorensen's (1985) second conclusion was not supported by the results of Marks (1980), although the differences in heritabilities were not significant. When chickens were fed a lower energy diet, estimated heritabilities of weights at six and eight weeks of age were about .1 lower.

Further support for Sorensen's (1985) third conclusion has been presented by Boyer et al. (1963) and by Mérat and Prod'homme (1969). In their studies, several strains were reported to have had higher eight-week weight heritabilities when fed high nutrient density rations. Becker and Berg (1960) selected lines of White Plymouth Rock chickens through two generations for divergent eight-week weight on high and low nutrient density diets. No difference in realized heritability was apparent when selection was for high weight; however, heritability was higher (.42 vs. .25) when selecting for low weight if a high nutrient density ration was fed.

There is other evidence of nutritional influences on genetic variation of growth. Marks (1987) selected Athens-Canadian randombred normal and dwarf chickens for high and low eight-week weight based on performance when fed either normal (.4 percent) or high (1.6 percent) dietary salt levels. Selection differentials, gains, and realized heritabilities were higher when chickens were fed the normal diet. In another study, strains of Athens-Canadian randombred chickens were selected divergently through three generations for three-week weight after being fed selenium deficient diets (Cunningham et al., 1987). The asymmetrical response was greater for low weight. Response was greater for females than for males and was greatest in the first generation. Realized cumulative heritabilities ranged from .15 to .39 indicating that genetic variation for dietary selenium requirement exists.

Not all results reported above agree; however, if population sizes and standard errors of heritabilities are considered, some discrepancies should be expected. In fact, given the resources used, the results obtained are relatively consistent.

Leg weakness. Crippling, or leg weakness, in broilers has a negative effect on broiler production efficiency. In broiler flocks, this defect may not kill the broiler; however, in advanced stages it interferes with normal feeding and growth, and gives rise to condemnation of carcasses and of carcass parts. Moreover, broiler parent flocks are also affected, giving rise to poor performance in terms of production and fertility of hatching eggs. The genetic aspects of several types of crippling in chickens are discussed in Chapter 33.

Rate of feathering. Siegel and Dunnington (1987) have recently reviewed the literature pertinent to feather cover, providing the basis for the following statements. In addition to protecting the skin and body, adequate feather cover has insulative qualities that aid in thermoregulation, especially in cooler climates. Feather growth requires nutrients, resulting in a compromise between utilization and conservation of resources. During growth, feather replacement follows a sequence of down, juvenile, and postjuvenile plumages. The following genetic information is known: feather growth is influenced by sex-linked as well as autosomal loci; sex-linked early vs. late feathering permits visual distinction of the sexes of crossbred chickens at hatching; heritability estimates generally have been moderate; during three generations of selection for an index of feather cover of early feathering chicks, additive genetic variation was evident; studies of correlated responses involving feathering rate are few. In one study, correlated responses in back feather cover following divergent selection for eight-week weight were inconsistent. Pym et al. (1984) noted that in lines segregating for the sex-linked rapid and slow feathering alleles, the slow allele was fixed by selection for feed consumption between five and nine weeks of age, but the fast allele was fixed by selection for either weight gain or feed efficiency between five and nine weeks of age.

REPRODUCTION IN RELATION TO GROWTH AND MEAT PRODUCTION

Efficient production of chicken meat requires not only rapid growth and high yield of quality meat from the chickens produced, but also large numbers of vigorous healthy chicks at hatching time. The latter requires a large number of settable eggs per dam, and adequate regular insemination of each of the hens by cockerels to ensure high rates of fertility and hatchability. Reviews of literature concerning reproductive complications associated with selection for broiler growth (Siegel and Dunnington, 1985), direct and correlated responses to selection for growth (Marks, 1985), and the genetics of ovulation efficiency (Foster, 1985), summarize the available genetic information regarding reproduction rather well.

Male reproduction. Few studies are available regarding genetic parameters of traits related to the role of the male in reproduction. However, heritabilities of semen volume, semen concentration, and semen motility have been reported. Siegel (1963b) reported estimates for these three traits based on sire and dam variance components measured in high and low weight lines of White Rock cockerels as .14, .01, and .29, respectively. Higher values were obtained by Soller et al. (1965) for these traits, measured in White Rock cockerels with estimates based on sire variance components (.41, .46, and .87, respectively). It is difficult to resolve the differences between studies in estimates unless differences in environment associated with geographical location are involved. In both studies, the estimate for semen motility is at least twice as large as estimates for the other traits.

Female reproduction. In a review of the genetics of ovulation efficiency (Foster, 1985), the surviving hen's egg production was attributed to two major components, sexual maturity and rate of lay. Siegel and Dunnington (1985) named age, body weight, and body composition as factors determining onset of sexual maturity in meat-type pullets. Lines with high body weights attain sexual maturity soon after 155 days of age; lines with low body weight do not begin egg production until a critical body weight or body composition is attained. In fact, in extremely small lines, some females never commence egg production. Refer to Chapter 29 for further details.

Foster (1985) partitioned rate of lay into four components: initial rate of ovulation, decline in ovulation rate with age, proportions of ovulations resulting in ovipositions, and proportions of ovipositions producing normal eggs. In their review, Siegel and

Dunnington (1985) describe many observations relevant to the above mentioned components. It was mentioned earlier that selection for high juvenile body weight enhanced follicle development and ova production; however, fewer normal eggs are produced. The discrepancy has been shown by Hocking et al. (1987) to be attributable to several factors. There may be more numerous yellow follicles, and more frequent atresia of these follicles, especially when pullets are fed *ad libitum* during rearing. There may be a double hierarchy of yellow follicles of similar size, giving rise to the presence of ovulated follicles in the body cavity of such hens, and to simultaneous presence of two or more yolks in the oviduct associated with disruption of normal shell formation and production of fewer usable eggs (i.e. EODES). Older broiler breeder hens may have few or no developing yellow follicles, and there may be atresia of yellow follicles and continued occurrence of multiple ovulations. Many components and consequences of these phenomena have been discussed in earlier research and they are reviewed in full by Siegel and Dunnington (1985).

These phenomena are less severe in pullets that are fed on a restricted basis during rearing. In fact, restricted feeding of commercial broiler parent flocks during rearing to enhance normal egg production is standard practice. Hocking et al. (1987) stated that yellow follicle number was associated not with abdominal fat weight but with feed consumption. The tendency for current broiler strains to consume feed at or near gut capacity (Siegel and Dunnington, 1985) appears to be a cause of these problems.

For meat-type chick production, egg weight is also important. It has been shown in several studies to have a positive effect on broiler weight.

Heritability of female reproductive traits. Sexual maturity of heavy breed hens had average heritabilities based on sire, dam, and sire and dam variance components, and on dam-daughter regression of .39, .25, .32 and .31, respectively (Kinney, 1969). These values indicate that sexual maturity is moderately heritable.

Egg production, expressed in various forms, of heavy breed hens had averages of heritabilities of .36, .20, .25 and .21 based on sire, dam, and sire and dam variance components, and on dam-daughter regression (Kinney, 1969). Hence, egg production is only moderately heritable. Less information is available regarding components of egg production. Little is known genetically about causes of the atresia and multiple hierarchy of yellow follicles that appear to give rise to chronic internal layers and defective eggs that are part of the EODES syndrome. Siegel and Dunnington (1985) stated that the heritabilities of double-yolked and of extracalcified compressed-sided eggs are high, and the incidence of these can be changed by genetic selection. In addition, they are less frequent in comparable strains of dwarf hens.

The heritability of egg weight is high for heavy breed hens; it is .57, .65, .67, and .63 for early egg weight and .58, .54, .58, and .46 for mature egg weight for averages based on sire, dam, and sire and dam variance components, and on dam-daughter regression, respectively (Kinney, 1969).

Relationships among female reproductive traits. Marks (1985) has summarized the genetic correlations involving sexual maturity, egg production, and egg weight reported by Kinney (1969). Sexual maturity had genetic correlations of -.11 and .07 with egg production and egg weight, respectively; the corresponding correlation between egg production and egg weight was -.20. These values are too small to have major effects on the simultaneous genetic improvement of these traits. Their relations with growth rate have been reported earlier in this chapter. The effects of ten generations of selection for high feed consumption or for low feed conversion ratio measured between five and nine weeks of age have been reported by Pym (1985). Sexual maturity of the feed consumption line pullets was slightly delayed (9 days); that of the feed conversion line pullets was more severely delayed (23 days). Egg production of pullets was slightly depressed (20 eggs) for the feed conversion line and more severely depressed (62 eggs) for the feed consumption line. Siegel and Dunnington (1985) noted that, due to the influence of body size and composition on reproduction, these traits should be monitored during selection programs to improve feed efficiency or to reduce fatness.

Fertility and hatchability. The successful transfer of adequate normal viable spermatozoa to an oviduct suited to enhancing their survival is necessary for fertility (Siegel and Dunnington, 1985). Various fertility measures of meat-type chickens, such as percentage of fertile eggs, duration of fertility, and percentage of fertile hens, are positively correlated. Estimates of heritability of percentage hatchability are low, often .1 or less (Kinney, 1969). However, response to selection for duration of fertility has been demonstrated for both males (Ansah and Buckland, 1983) and females (McDaniel, 1983, as cited by Siegel and Dunnington, 1985). In the research of Ansah and Buckland (1983), realized heritability for duration of fertility following insemination of hens using previously frozen and thawed semen was .17.

Under natural mating, mating behavior becomes an important component of the fertilization process (Siegel and Dunnington,1985). Comparisons of males and females from lines selected for either high or low eight-week body weight revealed that fewer courts and matings occurred among high weight than low weight lines of chickens. Differences among males were attributed to differences in both libido and mating efficiency; courts and completed matings were three times as frequent for low line as for high line males. Females from the two lines mated to common types of males received similar numbers of courts; however, low line females experienced three times as many completed matings. In an experiment testing divergent selection for mating frequency, positive results were observed; however, no correlated responses in body weight were observed. Possibly asymmetry of correlations exists between these traits.

As with fertility, the role of genetics in determining hatchability is low relative to other factors. Kinney (1969) reported average heritabilities for hatchability of total eggs set and of fertile eggs set of .09 and .14 respectively, based mostly on studies involving heavy breeds. Ten generations of selection for weight gain, feed consumption, and feed conversion ratio between five and nine weeks of age depressed hatchability of all eggs set by 9.1, 7.6 and 2.7 percent respectively, relative to the unselected control strain (Pym, 1985). During 27 generations of divergent selection for eight-week body weight, hatchability of fertile eggs and total eggs set declined by .7 and .3 percent on average in the high line, and rose by .3 and .6 percent on average in the low line respectively per generation (Dunnington and Siegel, 1985). These results appear to be consistent with the increase in frequency of chromosomal aberrations in high weight strains reported earlier. Chromosomal aberrations cause embryonic mortality (Fechheimer, 1971) and reduce hatchability, especially types such as reciprocal translocations that can be perpetuated each generation. Refer to Chapter 19 for further details.

SUMMARY COMMENTS

Growth in chickens is definitely polygenic, with differences among breeds and strains, and heritabilities based on additive genetic effects around .4. Heritabilities based on the various methods of estimation indicate that estimates based on sire or sire and dam variance components are the best indicators of response to selection. Estimates based on dam variance components from nested mating designs overestimate realized heritabilities due to the combination of nonadditive genetic and maternal effects and the multiplication factor (4x) used to equate the dam component to the additive genetic variance.

Measures of growth, such as body weights and weight gains, are positively correlated with most measures of meat production, such as carcass and meat yields, weights of chemical components of the carcass, abdominal fat weight, and conformation and skeletal measures. Lower correlations are found for percentages of carcass fat and abdominal fat. In fact, weight gains after three to four weeks of broiler growth tend to be negatively correlated with percentage abdominal fat. Growth measures are also positively correlated with feed consumption and efficiency, ovulation rate and egg weight in females, and with incidence of chromosomal aberrations. Negative correlations exist between growth measures and male and female reproductive traits and fertility and hatchability of eggs. Consequently, breeders use specialized sire and dam lines for broiler production to minimize the effects of these negative relationships.

Breed and strain differences have been demonstrated for most traits associated with meat production; the Cornish breed and crosses have been consistently superior for carcass and meat yields. Most meat production traits are highly heritable (.5 or higher). However, values for carcass yield or evisceration loss are lower (about .3). With the exception of ash, heritabilities of chemical components of the carcass tend to be high (.4 or higher). Realized heritabilities for abdominal fatness have supported the high estimated values.

Carcass fat percentage is not only negatively correlated with other chemical components but it also differs relative to the other components in signs of correlation with other traits. Fatness has a negative genetic influence on feed efficiency. Fatness also has a negative phenotypic effect on reproduction; however, this can be overcome in commercial breeder and broiler parent flocks by restricted feeding during the rearing period.

The reproductive performance of meat-type chickens is inferior to that of egg-type chickens. Numbers of yellow follicles in the ovaries of hens are an exception. Increased numbers of yellow follicles appear to be responsible for greater follicular atresia, internal ovulations, and the deformed eggs associated with the erratic oviposition and defective egg syndrome (EODES). This syndrome appears to be correlated with feed consumption. The incidence of eggs associated with EODES is heritable and is markedly reduced in dwarf hens.

Fertility and hatchability of eggs from meat-type hens are low and these traits have low heritabilities. The increased incidence of chromosomal aberrations associated with increased growth rate appears to be responsible for at least part of the reduction in hatchability.

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Chapter 26. GENETICS OF GROWTH AND MEAT PRODUCTION IN TURKEYS

E.G. Buss

Research studies of turkeys began over sixty years ago. All of the early investigations were concerned with growth. Varieties were compared and crosses among varieties soon followed. Subsequently, strains of the most popular varieties, especially the Bronze and White, were compared. The differences in rates of growth were studied by making crosses of varieties and strains to produce F_1s and F_2s and backcrosses of F_1s to parental lines. The F_1s of the first crosses of varieties sometimes exceeded the body weight of the better parent, but F_1s from strain crosses almost always fell between the two parental strains.

Measurements of traits such as keel length, shank length, body depth, and breast width were added to some of the early studies. Eventually it was concluded the breast width was the most important of the four. A balance or ratio among the other traits was recommended. However, selection to improve breast conformation in early commercial turkey stocks was achieved by simple 'hands on' examination of the heaviest birds, according to personal communication from those who did the selecting for two major breeding companies. Strains of both these companies had attained superiority for body weight and conformation before pedigree breeding programs were initiated.

Reproductive traits such as egg number, fertility, hatchability of fertile eggs, egg weight, broodiness, and sexual maturity were considered during the second decade of research in turkeys. Such studies have continued to the present. Growth rate was often found to be negatively correlated with reproductive traits.

During the third decade, heritabilities were reported for growth rate and reproductive traits. Most studies continue to include estimates of heritability for traits being reported. There has been a wide range of values reported for traits such as body weight and reproduction. However, there has not been any research directed toward an understanding of the differences; suggestions are made, but they are not accompanied by appropriate data.

Some of the early research in turkeys included carcass evaluations, and several such studies were conducted during the fourth and fifth decades. Increased breast yield has been recognized as the most important variable associated with ever-increasing body weights. The yield of edible meat increases with increased body weight, irrespective of sex.

Research published on other traits influenced by body weight included efficient use of feed, livability, heart defects, and leg problems. Physiological measurements in turkeys with differences in rate of growth have been sparse. Reports on inbreeding effects on growth rate are inadequate for any conclusion regarding this breeding practice. Single-gene traits and their effects on weight gain have been reported throughout the years of investigations, and limiting effects on weight gain by mutant genes determining feather color in turkeys have been considered.

Selection experiments have been common during the last four decades, and most were carried out with mass selection for single quantitative traits. In recent years new directions for selection often originated in lines previously used for other objectives. The worth of some of the selection experiments appears to be rather limited. To have larger and specifically defined populations for selection experiments that may yield meaningful data on recognized problems, cooperative efforts with large commercial organizations are undoubtedly necessary for future research in turkeys.

CRITERIA FOR GROWTH

Appropriate factors and methods are essential for research into matters related to growth. Studies have included the rate of embryo development, the effect of egg size, and the association of body weights at various ages with final market ages. The major finding is that selection for increased body weight should pertain to the age at which turkeys are marketed.

The differences in body weight between the sexes are consistent; thus, data from most experiments have been reported separately for each of the sexes. The ratio of female weights to male weights among families and strains has been shown to vary over a rather large range, but the physiological reasons for such differences and the possible association of differences with reproductive traits have not been studied.

Age And Growth Rate

The rate of development during incubation, the influence of egg weight, and the rate of growth posthatching have been of interest since research in turkeys began. The effect of egg weight on growth is maximum at hatching, and it may be considered as a temporary environmental influence (Bray, 1965). Embryonic weights among four lines (one selected for increased weight at 12 weeks, one for increased weight at 24 weeks, a control line for these two selected lines, and a commercial strain cross) were found to be correlated with egg weight in three lines at 15 days, two lines at 18 days, and in all four lines at 21 and 24 days of incubation. Sire effects for embryonic weights at 18 days of incubation were highly significant. The rate of increase and decline in embryo weights varied among lines. Day-old weight is highly correlated with egg weight (Scott and Phillips, 1936), but there is no correlation between day-old weight and mature body weight (Funk, 1930; Scott and Phillips, 1936; Payne et al., 1957). The relative growth decreases from hatching to adult size (Asmundson, 1942; Asmundson and Pun, 1954b), as illustrated in Figure 26.1. Turkeys that make rapid gains early in life continue making greater gains during the following weeks (Funk, 1930; Asmundson and Pun, 1954a).

Maximum coefficient of variation occurs at four weeks of age (Funk, 1930). Correlation coefficients between body weight at early ages and 24-week body weight increase as age increases. Bumgardner and Shaffner (1954) reported correlations of .24 and .72 for body weights at four and 24 weeks of age, and 16 and 24 weeks of age in males, respectively, and .34 and .53 in females for the same age comparisons.

Economists, engineers, geneticists, managers, nutritionists and physiologists are interested in growth models that describe animal weight as a function of age. The Gompertz growth model has been found to fit the average weight and the 95 percent enveloping weight limits as a function of age. From the model, expected weight of a group of animals of the same strain at any given age can be determined within the limits of the model (Buffington et al., 1973).

The Gompertz equation which provided an excellent fit was in the form:

$$W = A \cdot \exp[-B \cdot \exp(-C \cdot t)]$$

where: W = weight at time t in kg, and t = turkey age in days. The parameters A, B and C were interpreted as:

A = asymptotic weight approached; that is, the weight at time $t = \infty$ in kg

$A \cdot \exp(-B)$ = weight at time $t = 0$ in kg

C = rate of exponential decay of the specific growth rate in days^{-1}

Values of the parameters A, B, and C in the Gompertz equation were presented for the best fitting Gompertz equation to the means of the weights of the entire flock, the weights of all the males, and the weights of all the females.

Sex And Growth Rate

Sex differences in rate of growth persist throughout the growing period, with the males growing faster than females (Asmundson and Lerner, 1940; Asmundson, 1942). Many authors have published data that support this finding without exception. The relation of mean female weight as a percent of male weight declines progressively from day-old to 28 weeks of age (Funk, 1930; Asmundson and Pun, 1954b; Johnson and Asmundson, 1957b) as illustrated in Figure 26.2. Males weighed about 20 and 60 percent of 24-week weight at eight and 16 weeks, respectively, while females weighed about 25 and 70 percent of 24-week weight at the same two ages (Abplanalp et al., 1963).

The percentage of total variation in 24-week-old turkeys accounted for by differences in sex was nearly 90 percent for length of shank, about 60 percent for body weight and length of keel, and less than ten percent for width of breast (Asmundson, 1948).

Families differ in the amount by which males outweigh females (Shaklee et al., 1952).

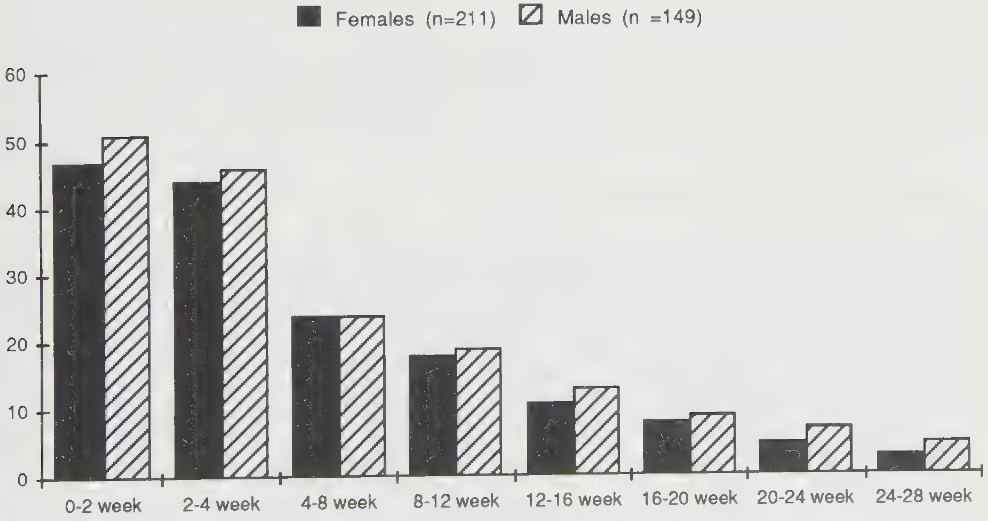

Figure 26.1. Rate of growth of turkeys in percent per week, 1946. (From Asmundson and Pun, 1954b).

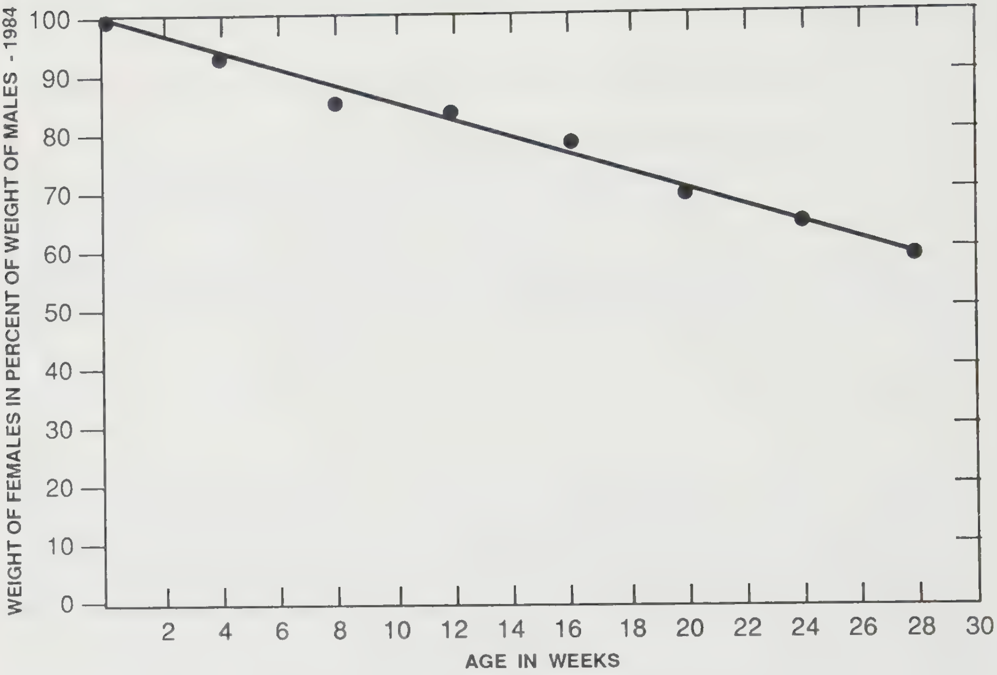

Figure 26.2. Weight of the female turkey as a percentage of the weight of the male. (From Asmundson and Pun, 1954b).

In families with the greatest sex difference, females weighed 51 percent of the weight of the males, while in families with the smallest weight difference, the female weights were 74 percent of those of the males. The average percentage for female weights of male weights was 62. The evidence suggested that heredity plays a role in the magnitude of differences between sexes at 24 weeks of age. Coefficients of variation as percentages of means for body weight were more variable for males than for females (12.2 vs. 9.71).

Variety Or Strain And Growth Rate

Early studies of the turkey focused on comparisons of varieties, including the Bronze, Bourbon Red, Black, Narragansett, White Holland, Slate, and Buff (Funk, 1930; Marsden and Knox, 1937; Jaap, 1938). Body weights differed among the unselected varieties. The heaviest males and females were found in the Bronze, and the lowest weights were in the Whites. There was little variation among the varieties for shank length, keel length, and body depth, and the differences were not consistent with differences in body weight. Investigations of crossbreeding by Asmundson (1942) included Broad-breasted Bronze, Black-winged Bronze, Black, Red, and Lilac; body weight differences existed among the varieties.

Intravarietal investigations were begun by Asmundson (1944), who used three strains of Bronze to study conformation. The males and females at 24 weeks of age weighed 6658, 7465, and 8298 g and 4418, 4793, and 5561 g, respectively, in the three strains. Since these early studies, much research has been based on differences among varieties and strains within varieties. Selection has contributed to large changes in several strains of Bronze and White. The rank order among strains for body weight and shank length measured at four-week intervals did not change from eight through 32 weeks of age (Asmundson and Lerner, 1942). Correlations between rate of growth and weight at 24 and 28 weeks of age showed that over half of the differences among strains for body weight are determined by differences in rate of growth between two and eight weeks of age (Asmundson and Pun, 1954a).

TRAITS ASSOCIATED WITH BODY WEIGHT

Early research in turkeys often included measurements of various traits such as keel length, shank length, body depth, and breast width. Extensive statistical analyses accompanied some of the studies, and several of these were published in Experiment Station bulletins. Various determinations of breast width were made, but eventually it was shown that there was no one measurement that was superior to others, because breast width increased at a constant rate.

Reproductive traits have been evaluated in experiments on selection for single quantitative traits such as body weight at 16 weeks of age or body weight at 24 weeks of age. Also, there have been selection experiments for increased egg production from which the effects on other traits, especially body weight, were assessed. The general summation of findings is that growth and reproductive traits are negatively correlated. However, it must be pointed out that there are exceptions. Perhaps the exceptions have not been anticipated and exploited as they might have been. Index selection for the two traits, growth and reproduction, has been reported with promising results.

Phenotypic Traits: Keel Length, Shank Length, Body Depth And Breast Width

Included in early studies of turkeys were measurements to determine the importance of phenotypic traits such as keel length, shank length, body depth, and breast width, in addition to body weight. Breast width determinations made by Jaap and Penquite (1938) suggested that satisfactory measurement may be made at a point 3.8 cm from the anterior end of the keel and on a line toward the insertion of the femur. Jaap et al. (1939) made linear measurements and expressed them as the ratio of the cube root of body weight. The values for keel length, shank length, and body depth were consistent, and the correlations between live and dressed weight were high. At 25 weeks of age, the shank length in males was 27 percent longer than in females at 21 weeks of age. Progeny of 'plump' birds had relatively shorter shanks, less body depth, and reached maximum weight earlier.

Measurements on three strains (one selected for egg production, one for good conformation and early development, and one considered to be the broad-breasted type) included length of left shank, length of keel, depth of body, width of breast, and weight at 24 weeks of age for both males and females (Asmundson, 1944). The three strains differed significantly in weight, length of keel, depth of body, and width of breast. The broad-breasted line was the heaviest and had the longest keel and widest breast, while the line selected for good conformation and early development was intermediate except for having the shortest keel. Differences in length of shank in the males were highly significant, while in the case of the females strain differences in shank length were of doubtful significance. The correlations between and within strains for length of shank and keel with width of breast were for the most part relatively low or insignificant. Coefficients of partial correlation for the linear measurements showed that length of shank was not correlated with length of keel, depth of body, or width of breast; length of keel was positively correlated with depth of breast in all groups; depth of body was either not correlated with width of breast or it was negatively correlated. The coefficients of correlation (*r*) based on total variance, when converted to coefficients of determination (r^2), showed that length of shank is influenced least, and width of breast is influenced most by variation in weight. Direct measurement of breast width at a definite distance above the lower surface of the keel is adequate, and it is the only measurement that needs to be made when increased width of breast is the objective. Bird (1948) considered the critical point for measuring breast width to coincide with 1/5th of the total depth from the crest of the keel to the spinal column. However, an excellent study comparing six points for measurement was conducted by Kinney et al. (1956). Correlation coefficients indicated that essentially the same relative rank was obtained when breast width was taken at 1/3rd or 1/4th of body depth as when breast width was measured at 3.8 or 4.4 cm from the anterior end of the keel. The results indicate that breast width increases at a constant rate, at least up to 1/3rd of body depth.

High correlations were obtained from logarithmic body weight and logarithmic shank length up to the time of cessation of shank growth; this indicates that general size factors govern the growth of the shank (Asmundson and Lerner, 1942). Analyses of heterauxesis indicate a uniform plan of growth from four to 16 weeks of age. The growth of the shank then decreases sharply after 16 weeks of age. Based on the length of shank at 16 weeks of age, the increase in length from 16 to 32 weeks was 3.5 percent. During the same period, body weight increased approximately 72 percent. The differences in proportion of length of bone to total weight of the bird appear to depend more on differences in growth of the entire body, as measured by body weight, than on the differences in growth in length of the bones.

The relationship of breast width and shank length was found to be that wider breasts were associated with shorter shanks (El-Ibiary, 1948). Furthermore, the coefficients of partial correlations, with variation in body weights eliminated, were both positive and negative. In males and females, respectively, the correlations for shank length with keel length were .43 and .46, breast width with shank length -.18 and -.39, and breast width with keel length .42 and -.33. In 28-week-old turkeys, El-Ibiary and Jull (1948) found coefficients of correlation of -.25, -.17, .07, and .07 between breast width and body depth, shank length, keel length, and body weight respectively. The *r* value between keel length and body depth and between keel length and shank length was .87 for both comparisons. When genetic variation was measured as the difference between the variance of the F_1 and F_2 generations and expressed as a percentage of the total variation, relatively large differences occurred between male and female percentages in shank length (17.4 and 69.2 percent), in keel length (0.0 and 27.3 percent), and in body weight (21.2 and 55.7 percent). Females exhibited considerably larger percentages of genetic variation in each of these three traits.

Lasley (1949) completed an exceptionally detailed analysis of data on three distinctly different lines of turkeys. Data from the three-year study were analyzed for measurements taken at ten, 20 and 28 weeks of age. Many single and partial coefficients of correlation were calculated. One conclusion was that correlations of weight with other traits decline with age. Also, the analysis suggested that correlations were higher for males than for females. For developing wider breasts, measurement of width of breast was far superior than other body measurements. Skeletal measurements were better indicators of weight than fleshing measurements during the first 12 weeks of age; then fleshing measures became increasingly important in determining weight. Maturity of the leg bones was reached before maturity of depth of body, while length of keel followed body depth in reaching maturity. No important difference was found in the ability of the three varieties to increase their weight at any given age.

Keel length and breast width were significantly correlated with the weight of breast muscle after variation due to body weight was removed (Johnson and Asmundson, 1957b). The analysis was based on data taken at eight, 16 and 24 weeks of age for 1161 Broad-breasted Bonze. The percent breast muscle increased from 15.7 at eight weeks to 19.2 at 24 weeks in males, and from 16.1 to 18.9 in females at the respective ages. Correlations between muscle weights and shank length were high at eight weeks of age, but they declined markedly by 24 weeks.

Important to studies of various aspects of the body is the repeatability of measurements. Analyses of measurements were carried out by Nestor and Chamberlain (1966). The repeatability of values measured by using a vernier caliper were .65, .31, .31, and .68 for shank length, keel length, breast width, and body depth, respectively. By using Bird's instrument (Bird, 1948), the estimates were higher for breast width and body depth (.44 and .98). The conclusion from this study was that two or more determinations of keel length and breast width should be made on each individual, while only one measurement is necessary for shank length and body depth.

Estimates of genetic correlations were calculated by both full-sib analysis and midparent offspring regression for body weight with shank length, keel length, body depth, and breast width at 24 weeks of age by Nestor et al. (1967). Data from a control population and three sublines, each selected for a single trait, were analyzed. Genetic correlations between body weight and body measurements were all large and positive. Shank length, keel length, and body depth were all highly correlated (above .52) in the control population. Genetic correlation estimates tended to be lower in the selected lines.

The same rank order for correlations of shank length, keel length, and breast width with body weight occurred in both males and females from Broad-breasted Bronze, Standardbred Bronze, Beltsville Small White, and the F_1 from Broad-breasted Bronze x Beltsville Small White (Kondra and Shoffner, 1955a). Body depth was less in the much smaller Beltsville Small White, but there were not any differences among the other three groups. It was concluded by the authors of this study that breeders will gain very little by taking into account body measurements other than body weight.

Reproductive Traits

Egg production and egg mass were found to be positively associated with body weight in both a large- and a small-bodied strain (Robel, 1981); however, linear regression curves for egg weight differed for the two strains. Payne et al. (1957) found that egg weight increased with increases in body weight; an increase in body weight of 230 g resulted in an increase of one g for egg weight. Nestor et al. (1972) found that the genetic correlations of body weight with 50 percent production, percent fertility, hatch of fertile eggs, and numbers of poults were -.05, -.21, -.40, and -.27, respectively. By grouping body weights of turkey hens, Parker and Harper (1950) found that neither the width of breast nor body weight of turkey hens within the limits studied had little or any relationship to the production of eggs or poults. Kondra and Shoffner (1955a) found that strains of similar body weight vary in egg number, fertility, and hatchability, and conversely, that strains varying greatly in body weight may produce similar numbers of eggs in which percent fertility and hatchability do not differ.

Hale (1957) concluded that broad-breasted males with a good balance of shank length, keel length and breast width can have 100 percent fertility, whereas males with wide breasts, very short keels and short shanks may be infertile. He suggested that shank length and keel length should be twice the width of the breast.

Other Traits

Feed efficiency. The relative efficiency in gains was greater in males than in females (Funk, 1943). The difference was highly significant after the turkeys were 16 weeks of age, and the difference increased with further age. Efficiency of gain was closely related to the rate of growth. Headley (1948) found feed conversion per unit of gain had an inverse relation to daily gain; the greater the gain, the smaller the amount of feed consumed per unit of gain. Furthermore, the rate of daily gain depended on feed consumption, regardless of variety or sex. Lucas et al. (1949) also concluded from a study of six strains representing four varieties that the largest strain was the most efficient and the smallest the least efficient in utilizing feed. Feed efficiency was best for the heaviest and poorest for the lightest of three strains at 12, 15 and 18 weeks of age (McCartney, 1952).

Although there is a great interest in feed efficiency, data showing differences for efficiency of feed utilization by turkeys of equal weights at a given age are not available.

Meat yields. Studies of the composition of turkeys as affected by age and sex showed that the percent dressed weight increased with age from 69 at 16 weeks of age to 76 at 32 weeks (Harshaw and Rector, 1940). Likewise, the percent breast muscle increased while percent leg muscle showed a downward trend with age. The percent skin increased from five to nine in both males and females. Percent protein in the breast muscle (24.6 percent for both males and females) and in the leg muscle (20.6 percent for males and 20.8 percent for females) did not change as weight increased from 3476 g at 16 weeks of age to 7928 g at 32 weeks in males, and from 2600 g to 4881 g in females during the same interval. Harshaw et al. (1943) conducted a similar study utilizing Broad-breasted Bronze, Standardbred Bronze, White Holland, and Beltsville White strains and varieties at 28 weeks of age. The body weight differences (from 11.2 to 6.8 kg for males and 7.1 to 4.1 kg for females) were not indicative of differences in percent breast muscle, leg muscle, total edible meat, skin, edible organs, internal fat, and external fat.

The amount of edible meat on eviscerated carcasses from medium-sized Broad-breasted Bronze and Beltsville Small Whites at 31 weeks of age varied with weight. Turkeys of any given dressed weight and at the same age had the same amount of edible meat (Headley, 1948).

Differences among 12 broad-breasted strains were significant for percent breast meat but not for thigh meat (Fry et al., 1962). At 22 weeks for females and 26 weeks for males, the percent breast, without bone but with skin, ranged from 31.3 to 34.0 for males and 29.4 to 31.4 for females, with the breast yield being positively correlated with eviscerated weight in males. Hartung and Froning (1968) conducted a similar study using Broad-breasted Bronze, Broad-breasted White and Beltsville Small White. The turkeys were sacrificed at biweekly intervals from 14 through 24 weeks of age for females and from 14 through 28 weeks for males. The percent of eviscerated weight for meat and skin did not coincide with carcass weight. There were no differences for percent bone among the strains of females; however, males in two strains did differ significantly as body weight increased with age. The percent of meat increased from 61 to 66 in the males, while the percent of meat in females remained at 61. Conversely, bone decreased by ten percent in males and five percent in females as body weight increased. Skin increased by at least five percent in both males and females; at 28 and 24 weeks of age the percent skin was 12.3 and 12.5, respectively.

Among 13 strains of turkeys, percent total meat ranged from 68.1 to 72.9, breast meat from 29.0 to 34.5, and dark meat from 25.2 to 27.4 (Dobson, 1969). Total meat, and breast and dark meat yields were not consistent with body weights, and there was very little difference among strains for percent dark meat; therefore, increased body weight was accompanied by an increase in percent breast meat. The percent skin did not correspond to body weight. Four strains differing in body weight also had differences in yield of breast meat, and the yield differences were associated positively with 18-week body weights (Orr et al., 1974). Significant increases in growth rate of two strains during a 10-year period resulted in a greater proportion of breast and thigh meat (Moran, 1981).

Boneless and skinless breast and thigh yields varied among 12 strains (Taylor and Fry, 1967). Percent breast in males ranged from 23.4 to 29.7, and in females from 22.8 to 28.9. Percent thigh in males ranged from 12.9 to 14.1, and in females from 13.3 to 14.8. Eviscerated carcass weights were not indicative of either breast or thigh meat yields in either sex at ages of 22 weeks for females and 26 weeks for males. Among 15 strains in a random sample test, skinless breast yields from males ranged from 32.0 to 39.9 percent, whereas thigh meat ranged from 13.3 to 14.3 percent of dressed carcass weight (MacNeil, 1969). Percent total skin ranged from 5.1 to 10.4 percent of carcass weight. The differences were not associated with differences in carcass weights. Breast yields from females ranged from 27.9 to 38.9 percent of carcass weight and the carcass weights for these two extremes were identical. Thigh meat ranged from 13.9 to 15.2, and total skin from 5.4 to 9.2 percent of carcass weight, and the differences among strains were not associated with carcass weight.

Differences among six strains of Broad-breasted Bronze for tenderness of both breast and thigh muscle were not significant, but the difference between sexes was significant, the females having lower shear values at 26 weeks of age (Goodwin, 1966). Neither strain nor sex was consistently related to flavor, tenderness, and juiciness of roasted carcasses (Carlson et al., 1962).

Abdominal fat. The quantity of abdominal fat was measured in three heavy-weight lines and in a control population of Broad-breasted Whites (Bacon et al., 1986). The large-bodied turkeys did not differ in total abdominal fat, either as actual weight or as percent of live body weight. Furthermore, carcass ash, protein, fat, and dry matter did not differ among the three heavy strains. The lighter weight control line had less abdominal and total carcass fat, and less dry matter, but the ash and protein percentages were higher. The females had more than double the amount of fat found in the males; the same relationship was found for percent of body weight. Percent ash and percent protein were higher in the males. The genetic correlation between lines for body weight and weight of the abdominal fat pad was found to be .39. The correlations of body weight with percent ash, protein, total carcass fat, and dry matter were essentially zero, .09, -.05, -.01, and .04 respectively.

Chemical composition. The protein, fat, ash, and water content of the breast muscle, leg muscle, remaining edible portion, and total edible portion were determined in four varieties and strains that included Broad-breasted Bonze, Standardbred Bronze, White Holland, and Beltsville Small White (Harshaw et al., 1943). The fat content was lowest in the largest strain, and between sexes it was highest in the females. Protein, ash, and water content of total edible portions varied inversely with the fat content. Differences in maturity among strains at the time of analysis accounted for some of these differences.

Breast blisters. Among turkeys of 12 strains, those with breast blisters did not differ in body weight from those that did not have blisters (Rasplicka and Fry, 1963). Furthermore, breast blisters were not correlated with keel length or body depth. Turkeys with wide breasts and shallow bodies were more prone to have breast blisters than those with wide breasts and deep bodies.

Leg weakness. The problem of leg weakness, as seen by the outward bowing of one or both hocks, has been recognized since the turkey industry began expanding in the 1940s. The greater incidence among males has resulted in a general assumption that the extra body weight is the cause of weakening in the hocks. Buffington et al. (1975) studied two flocks of a medium-sized strain of Whites and found by using a contingency table that, within a sex, leg abnormalities were as frequent among the lighter weight turkeys as among the heavier ones. The total incidence among females was 4.8 percent and it was 16.1 percent among males. Lesions were first observed at 6-8 weeks of age and progressed to a more extreme condition in a gradual manner. In addition to sickled hocks, some had abnormal toes and twisted tibiae. The incidence of tibial dyschondroplasia did not differ among five lines of commercial turkeys (Walser et al., 1982). The highest incidence in the heaviest line was not significantly different from the lowest incidence in the line with the lowest weight. The length of the tibiotarsus did not correlate with the incidence of tibial dyschondroplasia. Gross deformities were noted at 14 weeks of age.

The incidence of hock abnormalities in males was found to be influenced by genetic differences (Johnson, 1956). The correlation among members of the same sire group was .77, and the incidence among 20 sire groups ranged from zero to 71 percent. There was no significant correspondence between genotype for this condition and the genotype for rapid growth rate to 12 weeks of age. No experimental evidence was found to show that rapid growth is the primary cause of any skeletal deformity (Riddell, 1981).

Bone growth and organ weights. Correlations of the weights and lengths of the body, systems, and organs in the turkey female were established by Latimer (1927). The correlation between net body weight and the weight of organs ranged from a high positive correlation of .87 for the eyeballs to a negative correlation of -.60 for the thymus. The weight of the heart, although only 0.5 percent of the total body weight, is correlated positively with kidney, liver, spleen and digestive tract weights. The hypophysis was slightly negatively correlated with body weight, but the thyroid, like the thymus, was negatively correlated (-.56). The weights of the bones in the leg were more closely related to body weight than were the bones of the wing. Latimer and Rosenbaum (1926) found that lengths of bones were more variable than weights, and that the tarsometatarsus had the lowest coefficient of variation compared with the humerus, radius, ulna, femur, and fibula.

Linear bone growth attained a plateau at about 16 weeks of age in females, and at 22 weeks in males (Sullivan and Al-Ubaidi, 1963). Tibiae were significantly longer in males than in females at ten weeks of age and thereafter. At 24 weeks of age, the amount of tibia ash in females was only 60 percent as much as in tibiae of males; the difference was apparent by ten weeks of age. Percent tibia ash, however, was four percent greater in females at ten weeks of age and two percent greater at 24 weeks.

Heart conditions. Sudden deaths of maturing turkeys have been frequent enough to attract research interest. A study of round hearts in four strains of Broad-breasted White turkeys from commercial breeders utilized 100 poults from each (Hunsacker, 1971). Recording of electrocardiograms was followed immediately by sacrificing the poults at three weeks of age. Mean body weight of poults with round hearts was lower than that of normals in each of the four strains. There was some suggestion that the depression was greater in males than in females. Strain differences for body weight did not appear to be indicative of the incidence of round heart, that ranged from three to 28 percent.

Three strains of Broad-breasted White differed significantly in the incidence of aortic rupture. This defect was not associated with the weights of the three strains. Two commercial strains had the same incidence, but the body weights were significantly different (Krista et al., 1969).

Blood pressure. Strains of Broad-breasted White selected for high and low blood pressure were crossed reciprocally (Shoffner et al., 1971). No indication of nonadditive variance, sex-linkage or maternal affects was observed. Six-week body weight showed little evidence of a correlated response to selection for hypertensive and hypotensive turkeys.

Growth hormone. The role of hormones that may be associated with growth has become a topic of research in recent years. Plasma growth hormone (GH) concentration during growth of Broad-breasted White turkeys was low at one week of age, measuring 32 and 30 ng/ml in females and males, respectively (Harvey et al., 1977). Levels peaked at four weeks in females and at seven weeks in males (185 and 142 ng/ml). At 17 weeks, the levels had decreased to 8.7 and 17.3 in females and males, respectively. Correlations between GH concentrations and body weight or age were inverse and significant; they were -.62 and -.69 for females and males, respectively.

Strains exhibiting a two-fold difference in body weight at 26 weeks of age showed a significant positive correlation between weekly percentage rate of growth and plasma GH concentration (Proudman and Wentworth, 1980). However, age and GH concentration were negatively correlated. Peak levels of the hormone were reached at two weeks of age and did not differ between the two strains. The rate of decline was significantly faster in the larger strain. Sex differences were noted only in the larger strain between the ages of nine and 18 weeks.

Pulsatile secretion pattern of GH in turkeys is unique for each individual (Shaw et al., 1987). The age at which peaks are achieved varies among individuals. The timing of peaks is not influenced by sex; however, higher peaks are achieved in males. Peaks per eight-hour period are nearly three times as frequent in four-week old vs. 14-week old turkeys, measuring 6.1 and 2.1, respectively.

Plasma GH in a line selected for 20 generations to increase rate of growth was significantly lower than in the control line (Vasilatos-Younken et al., 1988). However, within lines and over age, GH concentration was significantly correlated with weight (r = .83 to .93). Between three and ten weeks of age, the plasma level of GH was higher in males than in females, but the GH per unit of metabolic weight did not differ between sexes. Growth rate does not appear to be dependent on levels of plasma growth hormone.

Blood constituents. Blood glutathione and plasma cholinesterase levels in Large White and Auburn randombred turkeys were compared with levels in lines derived from each and selected for increased 12-week body weight. Both blood constituents at 13 weeks of age were significantly increased in the two lines selected for increased body weight. The correlations of glutathione and cholinesterase levels at 13 weeks with 12-week body weight were .67 and .48, respectively. However, male body weights at 24 weeks and female body weights at 22 weeks were not related to the levels of these biochemical constituents at 13 weeks of age (Mukherjee et al., 1970).

Blood group alleles. Broad-breasted Bronze with the $Ea\text{-}B^2$ blood type allele were consistently superior in 16-week body weight compared with those that lacked the B^2 allele. Consistent with this finding was that the frequency of the B^2 allele was markedly greater in the line selected for rapid rate of early growth than in the control line (Stevens and Asmundson, 1968). Thus, the B^2 allele may be considered as a marker for more rapidly growing turkeys. This terminology using the symbol *B* for a blood type should not be interpreted as having any association with the *B* symbol used for the locus which determines the black or bronze color in feathers.

Plumage color alleles. Allelic differences at six loci affecting feather pigmentation were studied for possible influence on body weight, egg number, semen volume, and sperm concentration (Nestor and Renner, 1979). Seven reciprocal backcrosses were made in which alleles at only one locus were segregating in a cross. Only the grey allele (c^g) at the *C* locus had any influence on the quantitative traits; it was associated with the production of more sperm when compared to the recessive white allele (*c*).

RESPONSES OF F_1s, F_2s, BACKCROSSES AND THREE-STRAIN CROSSES

The general conclusion from early research investigations is that body weight of turkeys is determined by an additive expression of several genes. Thus F_1s are generally intermediate in body weight to the two parental lines. Backcrosses have also exhibited the same nature of additive expression. Thus, crossing to improve body weight is not appropriate. Other phenotypic traits have responded to crossing in a similar manner. Likewise, crossing has not been demonstrated to be a reliable or predictable way of improving reproductive performance. When performance of the F_1 is above the average of the two parents, it generally occurs when one parent is noticeably inferior for the trait being considered.

Body Weight Alone

The early studies indicated that body weight of F_1s generally exceeded that of the heavier parent. Crosses of several varieties were made by Asmundson (1942). Blacks were mated to Black-winged Bronze, White, Red, and Lilac (*Sl/sl R/r*). The cross with Black-winged Bronze was made reciprocally. Except for the F_1s of Black x White that weighed less than the larger parental variety at 24 weeks of age, the F_1s exceeded the heavier of the parental varieties at 8, 16 and 24 weeks of age. Three strains of Broad-breasted Bronze and one strain of Standardbred Bronze were compared with their progeny from reciprocal and backcrosses produced from various combinations of the four strains (Kondra and Shoffner, 1955b). The 24-week body weight of F_1 males and females exceeded that of their parental lines, and the three-strain crosses weighed more than the F_1s. Two of four different backcrosses showed an increase over their respective F_1s and their two parental strains. The response of males was greater than that of females relative to the larger parental strain.

Males in all three different combinations of reciprocal crosses were slightly heavier when the female parent was from the heavier of the two strains. The females of reciprocal F_1s did not differ in a consistent manner.

More recent studies, however, showed that F_1s frequently failed to be as large as the heavier parent. Hayse and Moreng (1973) found a mixed response among F_1s from various crosses. Three strains of similar body weight at 23 weeks of age were mated in different combinations, and body weights of progeny at six, 12, 18 and 23 weeks were compared; some of the F_1s were below the smaller parent, most were intermediate, and some were above the larger parent. Conversely, Rooney (1957) did not find that body weight of F_1 males and females in four reciprocal crosses exceeded that of the larger parental strain. Varied responses were found by Jerome et al. (1960). Two reciprocal crosses were made between large White, medium-weight White and small White strains. At 14 weeks of age, the F_1s from males of the heaviest strain crossed with females of the smallest strain were intermediate, but males of the medium-weight strain crossed with females of the smallest strain produced F_1s that weighed more than the medium-weight strain. An eight-year study by Clark (1961) found that the average body weight of the F_1s only approached the body weight of the larger Broad-breasted Bronze parental strain. This study included Standardbred Bronze, Bourbon Red, Black, and Beltsville Small White, in addition to the Broad-breasted Bronze. The F_1 males tended to approach the weight of the large parent more than did the F_1 females. Friars et al. (1963) crossed reciprocally two strains of Broad-breasted Bronze, one a closed flock for eight generations and the other a large commercial flock established for 14 generations. Body weights of F_1s did not exceed the larger parental strain, and the reciprocal crosses did not differ from each other. MacNeil and Buss (1968) provided additional evidence that F_1s were intermediate to their respective parents. Reciprocal crosses from each of two pairs of commercial strains were used for this study. Nestor (1971b) affirmed the results showing that F_1s are intermediate to their respective parents. Four reciprocal and two additional strain crosses among seven strains ranging in body weight at 24 weeks of age from 8.0-13.5 kg were produced. F_1s in two of the reciprocals and the two other strain crosses did exceed the average of their respective parental strains, but they were not as heavy as the larger strain. The F_1s in the other two reciprocals weighed less than the average of their respective parental strains.

The crosses of varieties in the earlier years of research with turkeys indicated an advantage to producing F_1s; however, crosses made with heavier strains in more recent years do not support the earlier findings. Additive genetic inheritance for body weight is strongly indicated.

Body Weight And Other Phenotypic Traits

Shank length, keel length, and width of breast among F_1s and F_2s from matings of Broad-breasted Bronze and Beltsville Small White showed continuous variation (Knox and Marsden, 1944). There were no clear classes among the F_2s, suggesting quantitative inheritance.

The inheritance of breast width in two strains of Bronze was assessed by Asmundson (1945). Reciprocal crosses and backcrosses to the two parental strains led to the conclusions that autosomal genes are involved, and that at least some of the genes for breast width act independently of genes for body weight. Studies with the same two strains were extended further by Asmundson (1948). The F_1s and F_2s and progeny of backcrosses to the males of the parental strains were raised. Body weight, length of left shank, length of keel and width of breast were determined at 24 weeks of age. The F_1 progeny equalled or exceeded the body weight of the larger parent, but there was no difference in the progeny from reciprocal matings. The average weight of the F_2s was less than the average for the F_1s; the progeny from the backcrosses to the larger parent equalled the parental weight, while those from the other backcross were intermediate between F_1s and the smaller parent. The results for length of shank were, in general, similar to those for weight. The strains did not differ in length of keel, but the F_1 and backcross progeny generally had longer keels than the parental strains. The F_2s had shorter keels than the F_1s. Width of breast in the F_1 progeny was intermediate between that of the parental strains. The progeny from backcrosses were in each case intermediate between the F_1s and the parental strain. The width of breast for progeny varied with the width of breast of the parents. Significant differences in weight, length of shank, length of keel, and width of breast were found between full-sib families.

Reciprocal crosses, F_2s and backcrosses of two strains of Bronze, differing in weight at 24 weeks of age as well as in rate of growth from two to eight weeks, were made by Asmundson and Pun (1954a). The weights of F_1s tended to exceed those of the larger parent to 16 weeks of age after which F_1 males continued to weigh more, while the females weighed less than the larger parental strain. Backcrosses to the smaller parental strain and the F_2s were intermediate between the parental strains. The F_1s in reciprocal crosses did not differ in body weight; hence, neither sex linkage nor maternal effects were indicated.

Clark (1961) measured shank length, keel length, and body depth on males and females for eight years. With only one exception, and it was for males, the F_1s did not exceed the larger parent. Similarly, Nestor (1971b) did not find any increase in shank length, keel length, body depth, or breast width in either F_1 males or females from six crosses among seven strains; reciprocal crosses were made in four of the six strain crosses.

Percent breast meat and percent thigh meat from four reciprocal crosses, two F_1s from each of two pairs of strains, were intermediate with respect to their respective parental lines (MacNeil and Buss, 1968).

Nine pure strains, strain and variety crosses, backcrosses and three-strain crosses assessed at 4, 8, 16 and 24 weeks of age showed that general combining ability was much more important for body conformation than was specific combining ability (McCartney and Chamberlain, 1961).

Body Weight And Reproductive Traits

Using natural matings, percent fertility of reciprocal F_1s was compared with that of Small White and Broad-breasted Bronze parental varieties. The percent fertility of the F_1s did not exceed that of the higher parent, the Small White, but it was significantly higher than the poorer parental variety, the Broad-breasted Bronze. Percent hatchability of the reciprocal F_1s was intermediate to the two parental varieties. Each F_1 had hatchability that was essentially the same as that of the variety of its dam (Walter and Hoffman, 1947).

Kondra and Shoffner (1955b) did not find that egg number, fertility, or hatch of fertile eggs was increased in F_1 females produced from various combinations of three strains of Broad-breasted Bronze and one strain of Standardbred Bronze. Three of the strain crosses showed a ten percent improvement in fertility, and an eight percent improvement in hatchability, while backcrosses did not show any increased response. Two strains of males and three strains of Broad-breasted Bronze females were crossed reciprocally by Rooney (1957). Hatch of fertile eggs in all four F_1s was less than that of the better parental strain. Reciprocal F_1s from Broad-breasted Bronze and Beltsville Small White did not have hatchability that exceeded the higher parent, the Bronze. However, fertility of the F_1 from the Bronze male x the White female exceeded the better parent, the White.

An eight-year study of crossbreeding, using pen matings, included Standard and Broad-breasted Bronze along with Bourbon Red, Black, and Beltsville Small White (Clark, 1961). Neither percent fertility nor hatch of fertile eggs was consistently affected in females produced by crossbreeding. It was concluded that the additional work and expense involved in maintaining two varieties of turkeys for crossbreeding purposes was not justified.

Friars et al. (1963) did not find differences between reciprocal crosses for reproductive performance of males and females. F_1 males produced from the crosses showed consistent advantage for fertility over the pure strains. A high degree of infertility in the females of the commercial strain resulted in considerable variation among the F_1 females. Hatchability was not affected by strain of males, but F_1 females expressed an advantage. The most pronounced effect seen in the F_1 females was their marked increase in number of eggs produced. Cramer (1975), however, did not find any beneficial results for either fertility or hatchability when large and small lines of turkeys were mated to produce crossbred progeny.

Crossing strains or lines does not appear to be an effective way of improving egg numbers, percent fertility, or percent hatch of fertile eggs when the performance in both strains or lines is at an acceptable level. When levels are unusually low in one strain or line, F_1s used as breeders may be expected to be considerably better than the poorer parent.

Body Weight And Other Traits

Meat yield. Kondra and Shoffner (1955b) found that meat yield per dam decreased in F_1s, while three-strain crosses yielded more. Backcrossing had an adverse effect on body weight. Hayse and Moreng (1973) made two crosses from three strains having different body weights. The combined breast and thigh meat yield was not significantly different from that of their respective parental lines.

Fat grades. Crossbreeding studies by Clark (1961) found the fat in F_1s to be about intermediate between that of the parents. The body weights of the F_1s were intermediate to those of their parental varieties.

Feed efficiency. Feed efficiency varied little between F_1s and their pure strain parents (Clark, 1961). This was not surprising because body weights were not increased by crossbreeding. These results were from an eight-year study that used four varieties, including Broad-breasted Bronze.

Livability. Livability for poults from Broad-breasted Bronze, Standardbred Bronze, Beltsville Small White and F_1s from Broad-breasted Bronze x Beltsville Small White was approximately 80 percent for the three heavier groups and 15 percent less for the Beltsville Small White. Livability was neither limited by large body size nor enhanced by crossbreeding (Kondra and Shoffner, 1955a).

RESPONSES TO SELECTION

Selection for single quantitative traits or selection by an index of two quantitative traits has resulted in changes. The magnitude of change varies with the design of the experiment. Most selection experiments have used mass selection. Thus, it is not possible to judge if family selection is a more efficient or a more effective method. It seems unfortunate that most of the more recent selection research has not utilized family selection since it was recommended from studies conducted in the earlier years. Since it is well established that selection effectively changes parameters of traits, it seems highly questionable that more such experiments can be justified. Emphasis on physiological differences in desirable and undesirable birds and strains may be much more useful in providing information that may be utilized by breeders when making selections.

Body Weight Alone

Correlation coefficients calculated for body weights between ages two, four, eight and 16 weeks and body weights at 24 weeks in pedigreed flocks of Broad-breasted Bronze, having 11 sires and 53 dams, and Beltsville Small White, having 11 sires and 45 dams, were not high except for the correlation between weights at 16 and 24 weeks (Bumgardner and Shaffner, 1954). These authors concluded that selection of breeders for superior body weight should be made at or near maturity.

The correct relative emphasis on different traits so as to maximize improvement in a breeding program was made by Cook et al. (1962). This important work used a selection index that included total egg production during the first 100 days of production, percent fertility of all eggs set, percent hatchability of fertile eggs, body weight, and breast width at six months of age. The index was essentially one of multiple regression in which regression coefficients are derived from a set of n simultaneous equations, where n is the number of independent variables used in arriving at the total score of the index. Estimates of certain genetic and phenotypic parameters are required and a net worth or relative economic value is needed for each trait in constructing a selection index. The aggregate genotype (H) of a bird was designated as : $H = a_1G_1 + a_2G_2 + \ldots + aiGi$, where Gi is the genetic or breeding value of the i^{th} character and ai is the economic worth of the same character. The selection index was defined as: $I = b_1x_1 + b_2x_2 + \ldots + bixi$, where b is the regression coefficient or relative weight for the phenotypic value x of the trait in the index. The b values which maximize the correlation between I and H are solutions to a set of simultaneous equations involving the genetic and phenotypic variances and covariances and the economic worth. It was emphasized that the number of traits included must be limited so that the index will be moderately simple. This selection index was applied to data collected during a 12-year period. Body weight at six months of age increased from 5248 g to 7387 g; 3043 birds from 509 dams and 67 sires were used. It was concluded that this index provides an opportunity for a positive change in each of the traits and that body weight is the trait that would show the greatest response. Valid application of the principle of index selection assumes that the statistics used in constructing the indexes are reasonable approximations of the biological parameters of the populations. Troublesome inconsistencies arise from large sampling errors associated with the estimates of parameters.

Selection at 12 and 24 weeks of age for increased body weight was carried out for four generations in two lines of turkeys by Johnson and Gowe (1962). The two lines were equal in growth rate at the beginning of the selection program. Five males per line were used in the first year and ten males were used in the succeeding three years. Pedigreed matings were made from 8-10 females per male, and 900-1100 progeny were produced each year. Twelve-week weight of the males in the fifth generation had increased 16.1 percent in the 12-week line and 7.6 percent in the 24-week line; the females increased 13.6 and 6.0 percent, respectively. Increases at 24 weeks in the 12-week and 24-week lines were 18.0 and 22.4 percent, respectively, and 17.5 and 15.9 percent, respectively, for the females. The changes were more pronounced in the males than in the females.

Selections from a commercial population to increase body weight at eight weeks of age, at 24 weeks of age, and by an index designed to increase body weight at eight weeks without increasing body weight at 24 weeks, were carried out for seven generations (Abplanalp et al., 1963). The results were that the line selected for eight-week weight was heaviest at that age and, similarly, the line selected for 24-week weight sustained increases and weighed most at 24 weeks. The index line was as heavy or slightly heavier at eight weeks than the 24-week line, but was smaller than both the eight-week and 24-week lines at 24 weeks. At 24 weeks, the females of the index line weighed slightly less than those of the control line, while males were slightly larger than the controls.

Single-trait lines were developed by selection for egg numbers, eight-week weight, and 24-week weight from a randombred control population. Selection was carried out for four generations (McCartney et al., 1968). The numbers of breeders used were clearly defined for this experiment. Forty-eight pairs of parents were used to reproduce the control, eight-week, and 24-week lines in the first two generations; numbers were reduced to 36 pairs in the last two generations. The egg line was reproduced by mating 48 sires to 96 dams in the first two generations, and then reduced to 36 pairs in the following generations. Full-sib and half-sib matings were avoided. The proportion of offspring selected as parents ranged from 14-21 percent and from 17-21 percent, respectively, in the eight-week and 24-week lines. Selection was effective in increasing both eight-week and 24-week body weight. Genetic increases in eight-week and 24-week body weight lines were accomplished by increases in body weight at other ages, including maturity. The eight-week body weights of the males increased by 468 g in the eight-week line and by 313 g in the 24-week line, and the 24-week body weights of the males increased 1326 g in the eight-week line and 1839 g in the 24-week line. Increases in the females, although less, were similar in nature. Body weight at 24 weeks in the egg line decreased by 227 g and 272 g in the males and females, respectively.

Two generations of random mating preceded selection of a control line and a line bred for body weight at 12 weeks of age in each of two commercial varieties (Mukherjee and Friars, 1970). The lines consisted of 24 males and 48 females in each variety. The standard selection differentials per generation were 0.61 for one variety and 0.70 for the other variety. Significant positive regressions of the differences (selected - control) in means for body weight of males at 24 weeks of age and of females at 12 and 22 weeks on generations occurred in both varieties.

A large-bodied randombred population constituted the foundation for the development of two lines, one mass selected for increased 16-week body weight and the other mass selected by means of an index for both increased 16-week body weight and egg production (Nestor, 1977). The randombred population was continued as the control during nine generations of selection. Heritabilities, variances and covariances were used to construct the selection index used in the first two generations. The actual index values used for both male and female progeny produced in the later generations of selection were equal to the dam's egg production plus six times the 16-week body weight. Thirty-six pairs of parents were used in each generation for each line. The percent of progeny selected at 24 weeks of age from the first four weekly hatches averaged 24 and 21 for the 16-week weight and the index lines, respectively. Highly significant gains of .19 and .16 kg per generation of selection were observed for males and females, respectively, for 16-week body weight in the 16-week weight line, whereas gains in the index line at the age of 16 weeks were about a fourth as much in males and half as much in females compared to the respective gains in the 16-week weight line. Genetic increases in the 16-week body weight line at 16 weeks of age also resulted in significant increases in body weight at eight and 24 weeks of age. The 16-week index line, however, did not show a trend for change in eight-week body weight; the 24-week body weight did increase, but less at this age than in the 16-week weight line. Simultaneous selection for increased 16-week body weight and 180-day egg production resulted in increases for both traits. The linear regression coefficient of responses on generation of simultaneous selection was +0.11 and +0.08 for body weight at 16 weeks for males and females, respectively, and it was +0.7 for egg production.

Pooled reciprocal crosses of two commercial strains were used to establish a randombred population that served as a control; a line was selected from this randombred for increased body weight at 16 weeks of age (Nestor, 1984). The average body weight of the base population was close to the average of all large entries in the performance tests of the same year. The selected line was established by mass selection, and then 36 parental pairs were used in each of the following 16 generations. Four weekly hatches were used to reproduce the line in the early generations, but later more hatches were required to produce the desired number of progeny. The percentage of progeny selected averaged 28 percent for both sexes for all generations. Based on linear regression coefficients, the gains per generation in 16-week body weight were .15 and .13 kg, respectively, for males and females; the actual selection differential was .54 kg for sexes combined over all generations. Body weight gains in males at other ages (eight, 20 and 24 weeks) were .04, .12, and .20 kg per generation, while in females at the same ages, the gains were .04, .11, and .20 kg per generation. The large changes in body weight at eight, 20 and 24 weeks of age and at maturity in the selected 16-week weight line suggest that the genetic correlations between 16-week body weight and weights at all other ages measured are large and positive (Figure 26.3).

Data from two flocks of Broad-breasted Whites were studied for inbreeding effects during two generations (Cahaner et al., 1980). In seven male lines from a commercial breeder, an increase of ten percent inbreeding was associated with a decrease of 210 g body weight at 17 weeks of age; in eight female lines at the same age, the decrease was only 130 g. Thus, the effects were less in female lines, but there was more variation among the female lines than among the male lines.

Genetic correlation estimates among body weights at eight, 16, and 24 weeks were determined by both full-sib analysis and midparent offspring regression (Nestor et al., 1967). The combined values by full-sib analysis for the control population and three sublines were .83, .65, and .89 for 8x16, 8x24, and 16x24 weeks, respectively; the values for the same three age comparisons by regression analysis were .80, .60, and .93, respectively. The combined genetic correlations of body weights determined by full-sib analysis at 8x16, 8x24, and 16x24 weeks were .83, .65, and .89, respectively. Midparent offspring regression yielded almost identical values.

Kassid and Coleman (1984) reviewed many papers concerning selection experiments to

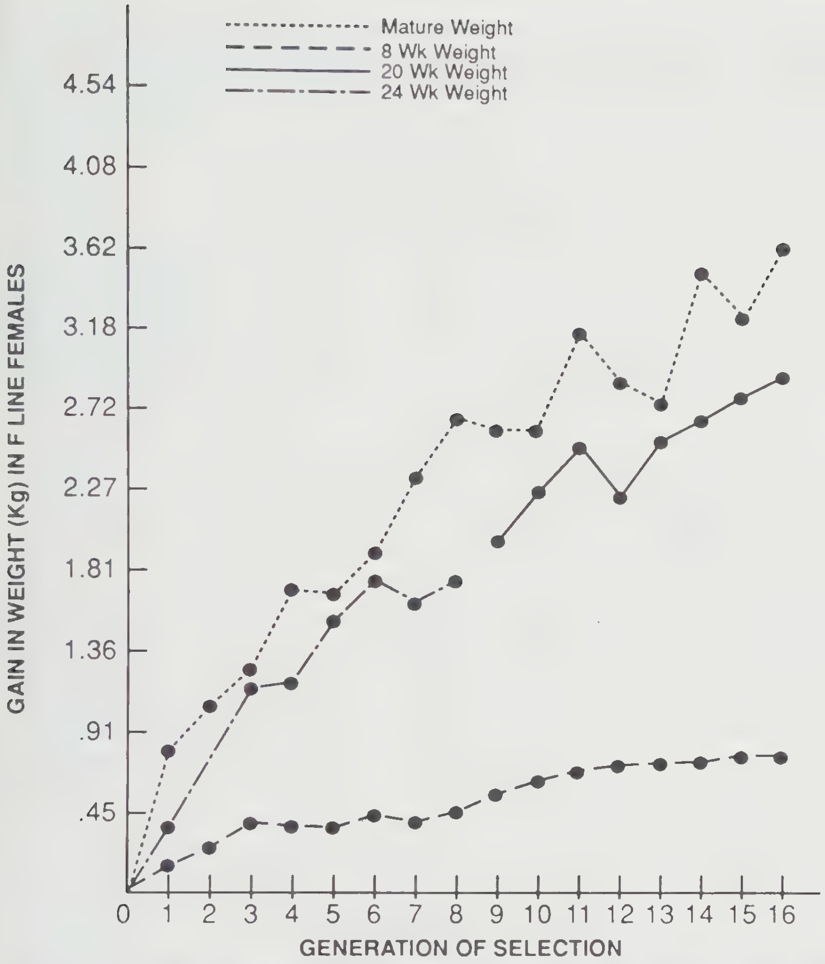

Figure 26.3. The effect of selection for increased 16-week body weight on body weight of females at eight weeks and 20 (Generations 9-16) or 24 (Generations 1-8) weeks of age and at maturity. (From Nestor, 1984).

study the inheritance of body weight, and they concluded that selection is an effective force in changing the means of populations over many generations either upward or downward. Asymmetrical responses have been more commonly observed than uniform responses in divergence between heavier and lighter lines; heavier lines gain more weight by plus selection than light lines lose by minus selection. Males gain or lose more weight than females when divergent selection is applied.

Body Weight And Other Phenotypic Traits

An analysis of parent lines of Broad-breasted Bronze and Beltsville Small White, and of their F_1s and F_2s, for body weight and correlations with keel length, shank length, body depth, and breast width led El-Ibiary and Jull (1948) to conclude that it is possible to establish strains differing significantly in skeletal proportions and plumpness of breast by selecting breeders on a progeny-test basis.

Selection in a flock of Broad-breasted Bronze over a period of 11 years was based primarily on body weight and increased breast fleshing at six months of age (Cook et al., 1962). Along with an increase in body weight, breast width measured at 4.5 cm in depth and 4.0 cm back from the anterior of the keel increased from 5.8 to 8.6 cm.

A selection index was used to increase body weight (W), shank length (S), and breast width (B) by Shannon and Armour (1962). The index was (W) + 3(B) - (S) for the first two years, and then it was changed to (W) + 5(B) - 2(S). On the basis of this index, 25 percent of the females and 12.5 percent of the males were selected at 24 weeks of age. During five years of selection, body weight and breast width increased steadily, but shank length increased very little. The coefficient of variation for the three traits declined during the years of selection.

Three lines were selected to increase egg production, eight-week body weight, or 24-week body weight (Nestor et al., 1967). After four generations of selection, genetic correlations for body weight and body measurements at 24 weeks of age by both full-sib analysis and midparent offspring were calculated. The correlations for keel length, shank length, and body depth with body weight at 24 weeks were .68, .61, and .66, respectively, by full-sib analysis; breast width was lower at .40. Midparent offspring regression produced similar values. The combined genetic correlations of breast width with keel length, shank length, and body depth were .24, .09, and .08, respectively. Combined correlations of shank length with keel length and body depth were .44 and .60, respectively, and the combined correlation of keel length with body depth was .67. These correlations were determined by full-sib analysis and all were positive; those determined by midparent offspring regression were lower and some were negative. Additional analyses of the above eight-week and 24-week weight lines showed that all body conformation measurements at 24 weeks of age also increased in both lines (McCartney et al., 1968).

A strain of Broad-breasted Bronze that was selected for five generations primarily to increase reproductive performance did not show any material changes in keel length, shank length, body depth, or breast width (Krueger et al., 1972). However, seven generations of selection to increase egg production by Nestor (1971a) resulted in a reduction in keel length, shank length, body depth, and breast width.

Average estimates of genetic correlation in female turkeys from different studies were published by Arthur and Abplanalp (1975), as shown in Table 26.1. The average correlations for body weight with keel length, shank length, body depth, and breast width are .50 or more.

Body Weight And Reproductive Traits

Selection of Broad-breasted Bronze primarily for increased body weight and breast fleshing for 12 years resulted in no change for number of eggs, percent fertility, and percent hatch of fertile eggs (Cook et al., 1962). With an average of 254 females per year, variation in the reproduction traits varied from year to year. Genetic correlations between egg number, percent fertility and percent hatch of fertile eggs with body weight and breast width varied considerably from sire and dam components of variance and covariance.

Selection for increased body weight at eight weeks of age in one line, at 24 weeks of age in a second line, and for increased number of eggs during a period of 180 days in a third

Table 26.1. Average estimates of genetic correlation in female turkeys in different studies. (Modified from Arthur and Abplanalp, 1975).

	Body weight					
Reference	Shank length	Keel length	Body depth	Breast width	Conformation	Egg number
Krueger et al. (1972)	.58	.79	.73	.79		
McCartney et al. (1968)	.45	.02	.50	.28		-.13[1]
Nestor et al. (1967)	.60[1]	.63[1]	.66[1]	.42[1]		
Cook et al. (1962)				.63[1]		-.05[1]
McCartney (1962)						.28
McCartney (1961)	.53	.56	.65	.53		
Johnson and Asmundson (1957a)	.70	.50	.67	.77		
Arthur and Abplanalp (1975)					.58	-.07
Average	.57	.50	.64	.57	.58	.03

	Egg number				
Reference	Conformation	Breast width	Keel length	Shank length	Body depth
McCartney et al. (1968)		.06	.04	.01	.25
Cook et al. (1962)		-.02[1]			
Arthur and Abplanalp (1975)	-.14				
Average	-.14	.02	.04	.01	.25

[1] Average of more than one estimate.

line, were carried out for four and five generations (McCartney et al., 1968). Selection for egg number increased production by 35 eggs to 120 days and 59 eggs to 180 days. At 120 days the eight-week line increased by ten eggs, and the 24-week line decreased by one egg. The control line produced 49 eggs in 120 days and 57 eggs in 180 days; thus, the egg number in the egg line doubled in five generations. Percent fertility decreased in all three selection lines; it was 91 percent in the control line during an eight-week period, while the eight-week, 24-week, and egg lines dropped 19, eight, and five percent, respectively. During the same period, percent hatch of fertile eggs dropped five, nine, and two percent below the 86 percent in the control line for the eight-week, 24-week, and egg lines, respectively. Consequently, poults per hen were fewer in the selected lines; the number was 23 for the control and five, seven, and seven less for the the eight-week, 24-week, and egg lines, respectively. The correlations of eight-week and 24-week body weights with egg production were -.04 and -.14, respectively.

Nine generations of mass selection were applied to two lines, one for increased 16-week body weight, and the other for increased 16-week weight and egg production using an index (Nestor, 1977). There was no significant trend in the difference between the two lines in 180-day egg production. This suggests that selection for increased egg production and body weight simultaneously by means of a selection index can result in increased weight without concomitant reduction in egg production, which occurs when selecting only for increased body weight. No significant correlations of 16-week body weight with egg weight, percent fertility, or percent hatch of fertile eggs were observed. Broodiness was not significantly correlated with body weight. An additional seven generations of selection did not change the association of body weight with fertility, hatch of fertile eggs, and broodiness (Nestor, 1984). The line selected for increased 16-week body weight had an increase in egg weight and a decrease in number of eggs produced in 180 days.

Seven male and eight female lines of Broad-breasted White were evaluated for inbreeding effects on egg number, egg weight, and hatch of fertile eggs (Cahaner et al., 1980). As inbreeding increased by ten percent, egg number and egg weight showed no consistent change, whereas hatchability decreased by 11 percent in the male lines. No trait was strongly or consistently affected by inbreeding in the female lines, but the traits were more variable than in male lines. The system of matings may have affected the results; full-sib and half-sib matings were avoided in the female lines, whereas they were allowed in male lines.

Lines selected for either length of clutch (C) or for a reduction in days lost from broodiness (B) lost weight; the selection was carried out for seven generations (Nestor, 1980). Percent fertility decreased by seven percent in the C line and by one percent in the B line; percent hatchability decreased by two percent and increased by one percent in the respective C and B lines. The C line had an increase of 8.8 eggs during a 180-day period, and the B line showed a loss of 19 days from broodiness. In the seventh generation, male weights in both lines and female weights in the B line had decreased significantly. The reduction in body weight of males and females in the C line was less than in the B line.

The number of eggs, weight of eggs, percent fertility, and hatch of fertile eggs were traits compared among three selected lines and a control (Ogasawara et al., 1963). One line was selected for heaviest body weight at eight weeks, one for heaviest weight at 24 weeks, and a third by an index of 5 (8-week weight) - (24-week weight). Each of the selected lines was reproduced with six males and five females per male; the control line had 12 males with three females per male. Natural matings were supplemented with artificial insemination at 50 percent production and again five weeks later during the first and the last four years of the experiment. Selection was continued for seven years. The results show that the average days to first egg was increased by nine and seven days in the eight-week and 24-week lines, respectively. From January 9 when supplemental lighting started until May 1, egg number increased to 60 in the eight-week line, while the 24-week and index lines produced only 49 eggs in comparison to 54 for the control. Egg weights were 89, 88, 84, and 86 g for the eight-week, 24-week, index, and control lines, respectively. Percent fertility, however, decreased to 62 in the eight-week and to 73 in the 24-week lines; it was 82 in the index and 86 in the control. The lines differed little from the control for hatch of fertile eggs; the percents were 73, 73, 77, and 76 for the eight-week, 24-week, index, and control lines, respectively. Of the four traits, percent fertility appeared to be affected most by selection for increased body weight, either at eight or at 24 weeks of age.

Selection to increase only 16-week body weight in one line and both 16-week body weight and egg number in another, with 48 sires and 96 dams per line, resulted in a loss in egg number by the body weight line and an increase in 180-day egg number in the index line (Nestor and Bachev, 1970). Twenty-four week body weights of both males and females increased in both lines during the seven years of selection.

After seven generations of selection to increase egg production, Nestor (1971a) realized a gain of 18 eggs in 84 days and 37 eggs in 180 days. During the generations of increasing egg number, body weight declined at both 16 and 24 weeks of age, whereas clutch length increased, broody periods decreased, days to first egg decreased, and egg weight decreased.

Body weights of females at 50 percent production in three lines were 3405, 4767, and 5539 g, and the numbers of defective eggs (broken, membrane only, soft-shelled, double yolks or slab-sided), were 19, 27, and 37 percent, respectively (Nestor and Bacon, 1972). The data were from 48 individually caged females per line. During the period of study, six females produced three eggs in 24-hour periods.

Selection to increase egg number for 19 generations showed a decrease in body weight, and selection to increase body weight at 16 weeks of age for 12 generations showed a decrease in egg number. Egg number for the egg line in 180 days of production was approximately double that of its control line (136 vs. 77), and in the body-weight line it was 48 eggs compared to 68 eggs produced by the control line (Nestor and Bacon, 1982).

After 11 generations of mass selection to increase 16-week body weight, tandem selection for increased 16-week body weight and for increased 120-day egg production was applied for four generations (Nestor, 1985a). Tandem selection resulted in a loss in body weight.

The 16-week body weight and the egg number lines were compared with their respective controls for responses to selection over many generations (Nestor and Bacon, 1986). In 24 generations of selection to increase egg number, the linear regression coefficient of response on generations in actual means was an increase of 3.9 eggs, a decrease of 0.001 kg in body weight, a decrease of 0.25 g in egg weight, and an increase of 0.27 kg in egg mass. In 18 generations of selection to increase body weight at 16 weeks of age, the linear regression coefficient of response on generation in actual means was 1.04 eggs, an increase of 0.37 g for egg weight, an increase of 0.16 kg for body weight, and a decrease of 0.74 kg in egg mass. Biological efficiency in each generation was defined as the ratio of egg mass:(body weight)$^{.75}$; it increased in the egg line and decreased in the weight line. Nestor (1985b) concluded that the consequences of improving growth had variable effects on egg production. The genetic correlations ranged from zero to strongly negative. The inconsistency of genetic correlations complicates the application of a selection index.

Body Weight And Other Traits

Abdominal fat. The amount of abdominal fat at 20 weeks of age increased during 14 generations of selection for increased 16-week body weight (Nestor, 1982). The increase in fat was both in actual amount and as a percentage of body weight. Fat as a percentage of body weight was double that of the control line. Both sexes showed the two-fold increase, but the fat percentages of females in the selected and control lines were nearly three times higher than those of males for the respective lines.

Leg problems. Selection for 16 generations to increase 16-week body weight resulted in an increase in the incidence of leg problems (Nestor, 1984).

Shank width. Shank width of a line selected for increased body weight at 16 weeks of age was greater than in the control line (Nestor et al., 1985). The difference was significant only for the males. In a line selected for increased shank width, the males increased in shank width by .19 mm and the females by .14 mm per generation. The males of the line selected for increased shank width had better walking ability than the line selected for body weight. The two selected lines had the same body weight.

Selection for increased shank width did not increase the weights of the tibiotarsus and femur. Measurements were made at 8, 16, and 20 weeks of age in the fourth generation of selection. The shank-width line and the line selected over 17 generations for increased body weight at 16 weeks had the same relative increase in weight from 8 to 16 weeks, and the same decline from 16 to 20 weeks of age (Nestor et al., 1987). Breast muscle, both absolute and as a percent of body weight, was less in the shank width line, but the lines did not differ in the relative amount of leg muscle at 16 and 20 weeks of age.

Plasma corticosterone. Plasma corticosterone was measured after cold stress of 4°C for four hours at four weeks of age in two lines, one selected for increased egg number and the other for increased body weight at 16 weeks of age (Nestor and Bacon, 1982). Compared to controls, plasma corticosterone level increased 15 percent in the egg production line and decreased 15 percent in the growth line. No sex difference in change was observed.

Ova development. The number of days needed for rapid development of ova in the ovary did not differ significantly between the lines selected for either increased egg number or increased body weight, the means being 11.8 and 12.0 days, respectively. The ova were significantly larger in the body weight line. These findings suggest that yolk production is not the limiting factor in egg production in meat-type turkeys (Nestor et al., 1970).

Four selected lines were compared with their respective controls for ovarian follicular development (Nestor et al., 1980). In the line selected for increased egg production, there was no significant change in number or total weight of follicles in rapid development at the conclusion of 180 days of production. In the line selected for longer length of clutch, the number of follicles in rapid development increased. In the line selected for days lost to broodiness, there was no change in the number of follicles developing. The total number of eggs increased for the line selected for increased length of clutch as well as for the line selected to have fewer days lost to broodiness.

Fat deposition. From base populations of ten males and 80 females from each of two commercial strains, selections with one male from each sire family and one female from each dam family were carried out for six generations to increase body weight at 12 weeks of age (Friars and Tiku, 1970). Controls were continued for each base population. Market grades were determined at 19 weeks of age for females and 23 weeks for males. Selection for early growth enhanced grade levels for back fat, breast fat, and fleshing, but not conformation. The faster growing strain of the two original commercial sources excelled in fleshing and conformation, but not in fat deposition.

Duration of fertility. Selection to increase 12-week body weight was assessed for any accompanying effect on duration of fertility (Friars, 1970). Two varieties were used for the three-year experiment. Duration changed from 13.6 to 12.5 days in one variety and from 17.8 to 13.6 days in the other variety; neither decrease was significant.

Livability. Percent mortality before 17 weeks of age averaged 6.1 and 5.7 for seven male lines in the two years that inbreeding was studied (Cahaner et al., 1980). The average percent mortality for eight female lines was 2.3 in one year and 2.4 in the other. An increase of ten percent in inbreeding corresponded with an increase of 1.4 percent in mortality.

Environmental influence. Lines from two varieties were selected for increased body weight at 12 weeks of age (Rodda and Friars, 1972). The differences in the selected lines over the respective controls at 12 weeks reflected differences that were found at 24 weeks of age, because there was no difference between selected and control lines nor between strains in growth between 12 and 24 weeks of age. The correlation between 12-week weights and 24-week weights was higher under favorable environmental conditions compared to adverse conditions. The genotype x environment interaction was significant.

HERITABILITY

It is common procedure to determine estimates of heritability with whatever data are available for nearly all traits studied. However, the usefulness of these estimates may be limited in view of the wide range of values reported for traits. Reasons for the wide range have been suggested, but the ideas have not been tested in research procedures. Until there are appropriate conditions established for obtaining estimates that are repeatable, it seems unreasonable to continue reporting heritability estimates.

Body Weight Alone

Abplanalp and Kosin (1952) used two methods of estimating heritability; one was based on the estimation of variance components and the other on offspring-dam regressions. The 4S/(A+D+S) was considered to give the best estimate of heritability, where A = the component of variance resulting from differences between full-sibs, D = the component of variance resulting from differences between groups of offspring from dams mated to the same sire, and S = the component of variance resulting from differences between groups of offspring from different sires. The highest value for body weight was 71 percent at 14 weeks of age. The heritabilities were consistently lower in the females, where the highest value was 39 percent at 14 weeks of age. The heritability estimates, 2(D+S), were considerably lower in Beltsville Whites, being 19 percent for males and 16 percent for females at 14 weeks of age. Strain differences were apparent.

Heritability estimates by full-sib analysis and by midparent offspring regression do not indicate that consistent differences occur (Nestor et al., 1967).

Heritability estimates based on variance components at 18 weeks of age were averaged over eight flocks (Arthur and Abplanalp, 1975); these averages along with comparable estimates for female turkeys reported in the literature are given in Table 26.2. The number of turkeys used in the study by Arthur and Abplanalp (1975) was 3991, and this exceeded the numbers in any of the previous studies. Hence, it is interesting to note that the heritability of body weight based on nearly 4000 turkeys in this study was .42 and that this is in good agreement with the unweighted average of .41 for all estimates shown in Table 26.2.

Nestor (1977) found heritability estimates for the 16-week body weight line to be .31 and .30 for males and females, respectively, after nine generations of selection. For the entire period of 16 generations of selection, the estimates were .29 for males and .24 for females (Nestor, 1984) (see Figure 26.3). Continued selection may result in a gradual lowering of heritability estimates. Nestor et al. (1967) used an average of four lines and found an estimate of 16-week body weight to be .42 by full-sib analysis and .50 by midparent offspring regression.

Table 26.2. Average heritability estimates in female turkeys in different studies. (Modified from Arthur and Abplanalp, 1975).

Reference	No.[3] sire groups	No.[3] females	Age in weeks at which body weight & conformation were measured	Body weight	Shank length	Keel length	Body depth	Breast-width
Krueger et al. (1972)	40	1384	21	.39	.39	.27	.32	.19
Mukherjee and Friars (1970) (Auburn)	20 to 36		22	.57[1]		.19[1]	.22[1]	.15[1]
Mukherjee and Friars (1970) (Large White)	20 to 36		22	.36[1]		.64[1]	.30[1]	.27[1]
McCartney et al. (1968)	168		24	.60				
Nestor et al. (1967)	672	3876	24	.39[1]	.44[1]	.46[1]	.40[1]	.12[1]
Cook et al. (1962)		2077	26	.45[1]				.24[1]
McCartney (1961)	184	1112	16	.56	.52	.62	.54	.26
Johnson and Asmundson (1957a)	12	163	16	.83	.73	.74	.57	.27
Kondra and Shoffner (1955a) (B.S. White)		246	24	.24	.43	-.12[2]	.70	.18
Kondra and Shoffner (1955a) (Bronze)		734	24	.50	.54	.33	.44	.32
McCartney (1955)	30	1030	16	.50				
Bumgardner and Shaffner (1954)	11	244	16	.44				
Goodman et al. (1954) (B.-B. Bronze)	14	1042	25	.29				
Goodman et al. (1954) (White Holland)	9	1025	25	.24				
MacLaury et al. (1954)		1331	26	.21				
Abplanalp and Kosin (1952) (B.-B. Bronze)	46	963	14	.29		.18	.40	.13
Abplanalp and Kosin (1952) (B.-B. White)	10	431	14	.16		.14	.21	.40
Arthur and Abplanalp (1975)	81	3991	18	.42				
Average			20.7	.41	.51	.36	.41	.23

[1] Average of more than one estimate.

[2] Used zero in obtaining average.

[3] If different numbers were involved for various traits, the number shown was the number available to estimate the heritability of body weight. Where there is a blank, the number was not reported.

Body Weight And Other Phenotypic Traits

Strain differences for heritability estimates derived by three methods for body measurements were determined by Abplanalp and Kosin (1952). The heritability of conformation was .28 at 18 weeks of age (Arthur and Abplanalp, 1975). Averages of estimates by several authors for keel length, body depth, and breast width were .36, .41, and .23, respectively, as shown in Table 26.2. In estimating heritability for breast width Nestor et al. (1967), McCartney (1961), Johnson and Asmundson (1957a), and Abplanalp and Kosin (1952) used the method of Bird (1948). Cook et al. (1962) also used Bird's device for part of their study. Mukherjee and Friars (1970) and Krueger et al. (1972) each used a specially designed caliper. Kondra and Shoffner (1955a) did not describe their method. No time trends were observed in heritability estimates for body weight and body measurements in lines selected for four generations.

CONCLUDING REMARKS

This chapter on genetics of growth and meat production of turkeys is divided into five sections: criteria for growth, traits associated with body weight, responses of F_1s, F_2s, backcrosses, and three-strain crosses, responses to selection, and heritability. An overview of the literature is presented as an introduction to the chapter, and more specific summations are offered at the beginning of each section. Major conclusions are as follows:

1. Individual bird selection for increased body weight has been effective.
2. Breast width is a better phenotypic trait than keel length, shank length, and body depth for determining desirable phenotypic carcass features.
3. Edible meat yield is increased as body weight increases.
4. The proportion of breast muscle increases as body weight increases.
5. Body weight at any specified age determines efficiency of feed utilization, and variation in feed efficiency for a specific weight at a given age has not been reported.
6. Leg weakness, primarily in males, is determined by genetic factors, and the deformities are not caused by increased body weight.
7. Crosses of strains have demonstrated that additive gene action determines body weight.
8. Selection experiments for increased body weight have been conducted mostly by mass matings.
9. F_2 populations from strain and line crosses have not been produced to assess the association or independence of body weight and reproductive traits.
10. Selection experiments for several traits have been initiated from lines already established by selection for another trait, and these subordinate selection experiments may have limited value.
11. Most selection experiments have been for only one trait.
12. Responses to selection have been reported for all traits studied.
13. Since many variables influence the outcome of selection experiments, additional ones on quantitative traits do not appear to be justified.
14. A wide range of heritability estimates has been reported for body weight as well as for associated traits.
15. The variation in heritability estimates suggests that research is needed for determining the number of birds and the mating system that will produce reliable and repeatable estimates of heritability.

16. Research in the future should focus on physiological and chemical parameters that may be useful for increasing body weight and meat production in the turkey.

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Chapter 27. GENETICS OF GROWTH AND MEAT PRODUCTION IN OTHER GALLIFORMS

H.L. Marks

Genetic variation observed for body weight and growth traits in chickens and turkeys also appears to be present in other galliforms. This chapter will review the topic of quantitative genetics of growth and selection for body weight in Japanese quail, guinea fowl and ring-necked pheasants.

GROWTH AND MEAT PRODUCTION IN JAPANESE QUAIL

Although a few commercial breeding programs have recently been initiated to develop Japanese quail lines with rapid rates of growth, little information is available regarding commercial breeding techniques and advances. Therefore selection responses for growth in Japanese quail will be viewed primarily from the results of experiments designed to provide an understanding of the inheritance of body weight and rate of gain.

Wilson et al. (1961) suggested Japanese quail as a pilot animal for more expensive experiments on chickens and turkeys. These authors noted that the rapid generation turnover should allow them to be an especially good pilot animal for genetic studies.

Heritability Of Body Traits

Heritability of body weight. Since heritability is an estimate of the ratio of the additive genetic variance to the total phenotypic variance, estimates would be expected to differ in diverse genotypes and under different environments. Therefore, discrepancies between heritability estimates for a particular trait should be considered the norm rather than the exception. The uniqueness of the population, the selection environment, and the length of prior selection, all can have a pronounced effect on the magnitude of the heritability estimate. Initial heritability estimates for body weight and growth rate in Japanese quail varied considerably. Collins et al. (1968), in replicate lines selected for three-week body weight, reported mean calculated heritabilities estimated from regression of offspring on mid-parent of .18 and .38, and realized heritabilities of .34 and .68. Marks and Lepore (1968) reported that in the first six generations of selection the realized heritability for four-week body weight was higher in a quail line selected on a high protein diet (.52) than on a low protein diet containing .2 percent thiouracil (.35). Calculated heritabilities (sire component of variance) for percentage growth rate from two to four weeks of age for quail lines under both lysine deficient and adequate environments were .31 and .36, respectively (Godfrey, 1968). Therefore, initial estimates for the heritability of body weight and growth in Japanese quail ranged from .18 to .68. Although this range indicates considerable variation, estimates are well within the range of heritabilities reported for body weight in chickens (Siegel, 1962; Kinney, 1969; Siegel and Dunnington, 1987).

Heritabilities of body weight in Japanese quail estimated by sire component analysis of

Table 27.1. Heritabilities for body weight traits in Japanese quail estimated by paternal (4S) and maternal (4D) half-sib correlations, and by parent-offspring regressions (b).

Method[1]	Line[2]	Age	Gen.	h^2	Reference
4S	F	2-4 wk[3]	1-10	.36	Godfrey (1968)
	D	2-4 wk	1-10	.31	Godfrey (1968)
	C	2-4 wk	1-10	.26	Godfrey (1968)
b_{mp}	S_1	3 wk	1-3	.18	Collins et al. (1968)
	S_2	3 wk	1-3	.38	Collins et al. (1968)
4S	P	4 wk	1-6	.56	Marks and Lepore (1968)
4D	P	4 wk	1-6	.63	Marks and Lepore (1968)
b_d	P	4 wk	1-6	.38	Marks and Lepore (1968)
b_s	P	4 wk	1-6	.29	Marks and Lepore (1968)
b_{mp}	P	4 wk	1-6	.45	Marks and Lepore (1968)
4S	T	4 wk	1-6	.49	Marks and Lepore (1968)
4D	T	4 wk	1-6	.38	Marks and Lepore (1968)
b_d	T	4 wk	1-6	.30	Marks and Lepore (1968)
b_s	T	4 wk	1-6	.61	Marks and Lepore (1968)
b_{mp}	T	4 wk	1-6	.38	Marks and Lepore (1968)
b_{dd}	C	5 wk	1	.44	Chahil and Johnson (1974)
b_{ds}	C	5 wk	1	.24	Chahil and Johnson (1974)
b_{sd}	C	sex. mat.	1	.63	Strong et al. (1978)
b_{ss}	C	58 d	1	.74	Sadjadi and Becker (1980)
b_{ds}	C	58 d	1	.69	Sadjadi and Becker (1980)
4S	P	2 wk	1-6	.51	Marks and Lepore (1968)
4D	P	2 wk	1-6	.53	Marks and Lepore (1968)
b_d	P	2 wk	1-6	.28	Marks and Lepore (1968)
b_s	P	2 wk	1-6	.20	Marks and Lepore (1968)
b_{mp}	P	2 wk	1-6	.25	Marks and Lepore (1968)
4S	T	2 wk	1-6	.51	Marks and Lepore (1968)
4D	T	2 wk	1-6	.50	Marks and Lepore (1968)
b_d	T	2 wk	1-6	.17	Marks and Lepore (1968)
b_s	T	2 wk	1-6	.48	Marks and Lepore (1968)
b_{mp}	T	2 wk	1-6	.25	Marks and Lepore (1968)
Average				.40	

[1] b_{mp} = regression offspring on midparent; b_d = regression offspring on dam; b_s = regression offspring on sire; b_{dd} = regression daughter on dam; b_{ss} = regression son on sire; b_{ds} = regression daughter on sire.

[2] C = control populations; all other lines selected for growth.

[3] Percentage growth rate from 2 to 4 weeks.

variance and regression analysis are shown in Table 27.1. These estimates were obtained from nonselected control populations or populations which have undergone only short-term selection for body weight. Although these estimates ranged from .17 to .74, the mean .40 of the 30 estimates is similar to the median heritability of .41 obtained from a summary of 176 published estimates for six-week to 12-week body weight in chickens (Siegel, 1962). These heritabilities for body weight in quail also fall within the range of estimates summarized by Kinney (1969).

Initial consideration of such a wide variation in heritability estimates for body weight is often perplexing. However, heritabilities must be considered as estimates from a given population under a given environment and at a given time following zero to n generations of selection. Sampling and measurement errors can also result in fluctuations of these estimates. A change in any of these conditions influences the heritability estimate.

Heritability estimates based on paternal (4S) and maternal (4D) half-sib correlations for body weight of male and female quail from 1-56 days of age are presented in Table 27.2 (Sefton and Siegel, 1974). Although these estimates were obtained in two different generations from the same population and under the same environment, there was considerable variability among estimates. These data further support the difficulty in obtaining a precise estimate of heritability for a given trait. The general trend in these estimates was an increase in the magnitude of paternal (4S) and a decrease of maternal (4D) estimates with increasing age. The usually large estimates (>1) based on maternal half-sib correlations at one day of age indicated a large maternal effect for measures of early body weight. Although there were several unrealistic values among the 72 estimates (Table 27.2), the means of estimates within sex, method of calculation and generation ranged from .29 to .62 with an overall mean of .43. This mean value is strikingly similar to the mean of .40 obtained from the summary of reports presented in Table 27.1.

Following 13 generations of selection in Japanese quail for increased weight gain (generations 1-5) and high six-week body weight (generations 6-13), relatively low realized heritabilities (<.25) were reported by Collins et al. (1970). Comparison of populations selected under three dietary environments (normal, diluted, and starvation) failed to disclose special adaptation of improved growth to diets.

Directional selection for growth at two ambient temperatures in Japanese quail resulted in a cumulative realized heritability estimate of .47 ± .15 for five-week body weight (Chahil et al., 1975). Data from this report indicated that selection for growth at high temperatures was effective. These data were contrary to reports of Marks and Lepore (1968) and Collins et al. (1970) that found lower heritabilities under stress environments.

In general, heritabilities for body weight in Japanese quail appear to be similar to those

Table 27.2. Heritability estimates x 10^{-2} ± standard errors based on paternal (4S) and maternal (4D) half-sib correlations for body weights of males and females by ages and generations. (From Sefton and Siegel, 1974).

	Males				Females			
	4S		4D		4S		4D	
	Generation							
Age, days	1 n=530	2 n=927	1 n=530	2 n=927	1 n=521	2 n=863	1 n=521	2 n=863
1	3±20	15±16	129±32	142±23	20±24	-5±16	159±33	182±27
7	36±28	26±11	37±22	12±14	25±18	28±13	56±24	49±17
14	11±17	14±11	68±26	45±16	27±18	39±14	48±23	25±15
21	-3±20	31±12	118±31	33±15	33±20	47±14	70±24	12±14
28	41±22	41±14	79±26	28±14	44±21	53±15	65±23	17±14
35	51±22	57±17	65±24	49±15	49±22	17± 7	68±23	-43±12
42	51±23	61±18	72±25	62±16	-21±19	62±17	148±32	31±14
49	39±21	62±18	65±25	50±16	42±19	46±15	40±21	25±15
56	49±21	51±18	49±23	76±17	46±20	46±16	34±21	43±16

for body weight in chickens (Siegel, 1962; Kinney, 1969). Therefore, Japanese quail should be an excellent pilot animal for studying the genetics of growth.

Heritability of body components. Kawahara and Saito (1976) estimated the heritability of various internal organ weights in Japanese quail. The organs measured were heart, lungs, liver, gizzard, intestines, pancreas, spleen, kidneys, gonads, oviduct, muscles, bones, and total body weight. The heritabilities for these internal organs ranged from .78 for bones to .17 for intestines, and showed no marked differences between male and female groups. In total body and muscle weight, however, males had higher heritabilities than females, and males had larger genetic variances for these traits than females. Total body weight and muscle weight were strongly correlated both phenotypically and genetically, whereas correlations among other organs were generally low.

Sato et al. (1982) reported heritabilities of eight-week body weight, carcass weight, and eviscerated carcass weight of .63, .61, and .51 respectively. Heritabilities of breast, thigh, and pectoral muscle weights ranged from .36 to .56. Heritabilities, based on sire and dam components, for eight-week body weight, breast weight, and thigh muscle weight were .67, .45, and .56 respectively (Sato et al., 1985). These authors also estimated the heritability of various skeletal parts and reported estimates of .41 to .84 for skeletal weight, .17 to .77 for skeletal length, .11 to .32 for skeletal width and .41 to .84 for skeletal height. It appears therefore, that the heritabilities of component body parts are similar to those for total body weight. These similarities would be expected due to part-whole relationships.

Maeda et al. (1982) calculated from sire and dam components of variance the heritability of the food conversion ratio from 4-6 weeks of age. Heritability estimates for food conversion ratio were .42 and .18 in males and females respectively. These estimates are within the range of heritabilities for food conversion and feed efficiency ratios in broilers (Wilson, 1969; Guill and Washburn, 1974).

Selection For Body Weight In Japanese Quail

Little consideration has been given to the selection history of populations from which estimates of heritability are obtained. Because selection responses are expected to be greater in early generations than in later generations (Lawrence, 1964), realized heritability estimates should differ in the same population following short- or long-term selection.

Influence of selection environments. The choice of environments under which selection is carried out represents a problem that is not completely solved. Early work of Falconer and Latyszewski (1952) suggested that poor environments (or stress environments) may be desirable because selection progress made under these environments was retained under good environments, whereas genetic gains realized under good environments were not carried over when animals were transferred to a poor environment. Because of the advanced technology employed in management, nutrition, and housing for poultry meat stocks, there is a consensus that selection should be performed in an environment similar to that in which birds are grown commercially. While this rationale appears to be basically sound, it must be viewed in both short-term and long-term contexts.

Marks and Lepore (1968) and Marks (1978, 1985, 1987) utilized Japanese quail to investigate the role of selection environment on both short-term and long-term selection responses for four-week body weight. The initial phases of this research involved two selected lines: line P was selected on an adequate 28 percent protein diet, whereas line T was selected on a low 20 percent protein diet containing .2 percent thiouracil (TU). A randombred control line (C) served as the base population for these two lines and also as a nonselected control during the duration of the selection study. To evaluate the concept of manipulating the selection environment, a third selected line (S) was established as a subline from the T line in generation 27. The S line was selected under a 28 percent protein diet containing TU.

Short- vs. long-term selection responses. The reports of Marks (1985, 1987) involving long-term selection for four-week body weight under different nutritional environments clearly indicate the importance of both the selection environment and the generations of selection on the magnitude of heritability for four-week body weight. Selection for increased four-week body weight in Japanese quail fed a 28 percent protein diet (P line) resulted in an increase in body weight from approximately 90 to 245 g (Figure 27.1) over a period of 60 generations of selection (Marks, 1985). The selection progress was greater during early than later generations (Table 27.3). The average gain (deviation from control) of 4.3 g per generation for generations 1 to 10 was twice the gain for generations 21 to 40 and three times that obtained from generations 41 to 60. Total response over 60 generations in four-week body weight in a quail line on a 20 percent protein diet containing .2 percent TU (T line) was not as great as the response observed in the P line (Table 27.3). However, selection responses (4.29 vs. 4.24 g) were similar for the two lines (Table 27.3) in generations 1 to 10. There was evidence that the decline in selection response was more rapid in the T line than in the P line and realized heritability estimates were consistently greater in the P line than in the T line (Table 27.4).

The influence of duration of selection on the magnitude of heritability estimates is clearly visible (Table 27.4). These data indicate that the realized heritabilities for four-week body weight were high (0.32 to 0.45) for generations 1 to 10, moderate (0.22 to 0.32) for generations 1 to 20, and small (0.00 to 0.17) for generations 21 to 40. These patterns are

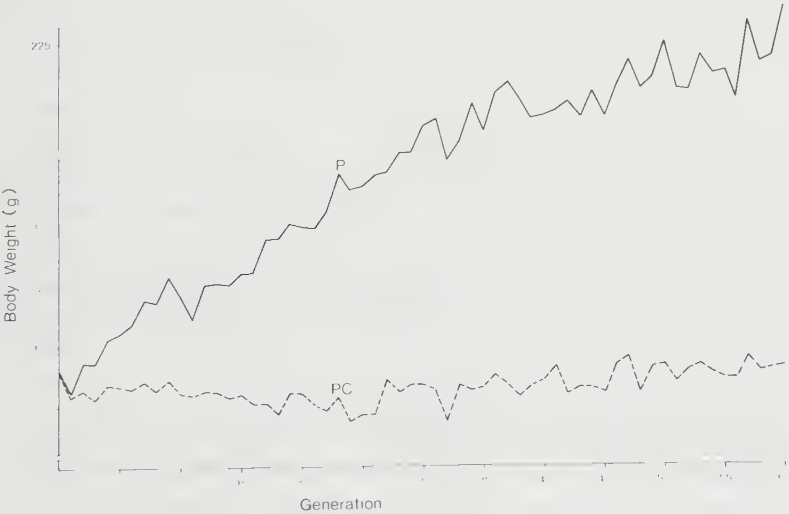

Figure 27.1. Mean body weight of P line and control line (PC) Japanese quail at four weeks of age through 60 generations. (From Marks, 1985).

Table 27.3. Regression of four-week body weight on generation number in selected and control Japanese quail lines. (From Marks, 1985).

	Selected		Control		Deviation from control	
Generation	P[1]	T[1]	P	T	P	T
1-10	4.22**	4.26**	-0.12	0.09	4.29**	4.24**
1-20	3.06**	2.53**	-0.28*	-0.29	3.34**	2.82**
21-40	2.44**	0.00	0.59*	-0.07	1.94**	0.09
41-60	1.63**	0.61	0.33	0.24	1.30**	0.37
1-60	2.30**	1.01**	0.18**	-0.19**	2.13**	1.20**

[1] P:28 percent dietary protein, T:20 percent dietary protein plus 0.2 percent thiouracil.
*P<.05; **P<.01.

Table 27.4. Heritability estimates for four-week body weight in selected lines of Japanese quail. (From Marks, 1985).

	P[1] line		T[1] line	
Generation	Realized[2]	Realized[3]	Realized	Realized
1-10	0.45	0.49±0.06	0.32	0.38±0.02
1-20	0.32	0.29±0.02	0.23	0.22±0.02
21-40	0.12	0.17±0.02	0.23	0.00±0.00
41-60	0.20	0.12±0.03	0.03	0.05±0.03
1-60	0.20	0.17±0.01	0.11	0.07±0.01

[1] P:28 percent dietary protein; T:20 percent dietary protein plus .2 percent thiouracil.
[2] Cumulative h^2=R/S.
[3] Regression.

in agreement with expectation from selection since it is assumed that the greatest progress will occur during initial stages of selection, to be followed by a gradual decline until response becomes negligible (Lawrence, 1964). Heritability estimates from early generations (0.32 to 0.45) are similar to those previously reported for quail (Table 27.1). More importantly, the magnitude of the heritability estimates for body weight in Japanese quail was observed to be very similar to those for body weight in chickens (Kinney, 1969; Siegel, 1962) suggesting that Japanese quail are an excellent model for genetic studies of growth in broiler chickens.

Influence of changing the selection environment. The short generation interval of Japanese quail (Wilson et al., 1961) has allowed them to be used as a model to investigate quantitative genetic theory, as well as a model to study the limits to selection. Continued selection in the P and T lines, and in a line S developed as a subline from the T line in generation 27, provides some insight into the complex behavior of long-term selection (Marks, 1989). Selection progress in the S line following modification of the selection environment (S line established by subjecting chicks from the T line to a 28 percent protein diet containing .2 percent TU) was greater than progress in either the P or T lines. Following correction for deviation from controls, the average increase per generation in four-week body weight of the S line from generation 27 to 70 was 1.24 g (Table 27.5). Corrected values for generations 27-40, 40-50, 50-60, and 60-70 were 1.19, 1.86, 2.45, and .88 g respectively. These data indicate that although progress was substantial from generation 27-70, during the last ten generations there was a decline in the magnitude of the selection response.

The appropriate evaluation of genetic response in the S line involves comparison with the T line from which it was originally derived in generation 27. A review of four-week body weight in the T line from generation 27-40 indicates that little if any increase occurred in body weights during this period, prompting Marks (1978) to suggest an apparent plateau in four-week body weight for the T line for generations 20-40 (Figure 27.2). The lack of response in the T line during this period is highlighted by a regression coefficient (of population mean on generation number) of -.02 g (Table 27.5). The regression coefficients of mean body weight on generation number for four-week body weight in the T line increased after generation 40. However, these coefficients were less than half the magnitude of corresponding S line coefficients prior to generation 60 (Table 27.5). Regression of mean four-week body weights on generation number for generations 27-70 of P line quail resulted in a corrected regression coefficient of 1.14 g. Comparison of regression coefficients between the P and S lines for generations 50-60 and 60-70 indicate that progress was greater in the S than in the P line during this period (Table 27.5).

Selection differentials and heritabilities under different selection environments. The greater response in S line quail than in T line quail following their establishment in generation 27 as a subline of the T line indicates that changing selection environments during a plateau may offer an effective way to disrupt the plateau and enable further genetic progress in quantitative traits. However, the effective (weighted) selection differentials in the S line were significantly (P<.05) smaller than selection differentials in the P and T lines which may have long-term ramifications. Realized heritability estimates in the S line were greater than comparable estimates in the T line except for generations 60-70 (Table 27.6). The S line estimates (.14 and .12) across generations (28-70) were more than twice as large as T line estimates (.05 and .06). In addition, the heritability estimates

Table 27.5. Regressions of four-week body weight (g) ± standard errors of selected (P, S and T) and nonselected (PC, SC and TC) quail lines on generation number. (Adapted from Marks, 1989).

		Generation				
		27-40	40-50	50-60	60-70	27-70
Selected	P	1.84**±.55	2.77±.60	1.87±1.21	-.91±.65	1.37**±.11
	T	.56±.65	1.09±.70	1.47±1.22	1.93±1.25	.89±.13
	S	1.94**±.32	2.22*±.73	2.84*±.96	-.05±1.25	1.40**±.11
Control	PC	.10±.34	.34±.49	.21±.32	-.24±1.25	.23**±.05
	TC	.60±.39	.38±.32	.34±.45	-.57±.44	.14*±.06
	SC	.75±.35	.36±.35	.39±.30	-.93±.47	.16**±.06
Deviation	P-PC	1.93**±.52	2.12**±.57	1.66±.99	-.68±.63	1.14**±.10
	T-TC	-.02±.29	.72±.52	1.13±.98	2.50*±.95	.74**±.10
	S-SC	1.19*±.48	1.86**±.47	2.45**±.73	.88±.90	1.24**±.10

*P<.05; **P<.01.

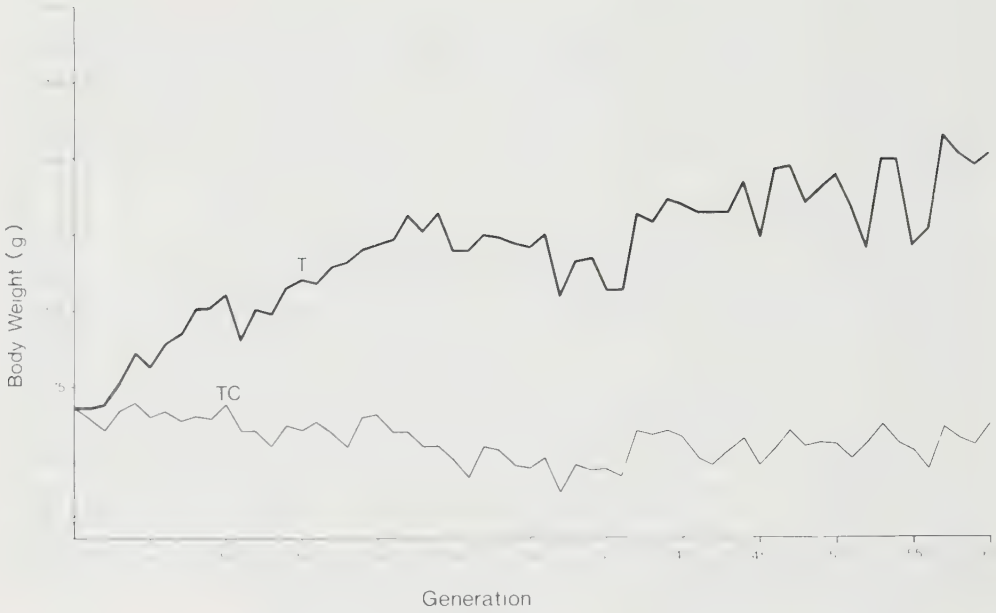

Figure 27.2. Four-week body weight of T line and control (TC) quail by generation.

over the 43 generation period were greater in the S line than in the P line (.14 and .12 vs. .10 and .10 respectively). Although numerically these differences were small, they actually represent a 20 to 40 percent difference. In general, there was a gradual decrease in the magnitude of heritability estimates in the S and P lines across generations (Table 27.6). These patterns are in agreement with patterns observed in the P line for generations 1-40 (Marks, 1978) and further support the general concept of diminishing responses to selection across generations (Lawrence, 1964).

The lack of a detectable selection response in the T line for an extended period (approximately 40 generations) followed by a resurgence in selection progress somewhere around generation 50 (Figure 27.2) is more difficult to explain. Jones et al. (1968) reported irregular response patterns in long-term selection for bristle number in *Drosophila* and reviewed previous studies in which 'waves of response' were observed. Yoo (1980) working with abdominal bristle number in replicate lines of *Drosophila melanogaster* reported periods of accelerated responses with intervening cessation periods. Roberts (1966) working with the mouse reported that a large mouse line showed a sharp increase in weight after remaining at an apparent limit for 20 generations. These data indicate that although disruption of a plateau is possible as a result of some unexplained event at a random point in time, the procedure of changing the selection environment to disrupt a plateau may offer a more controllable approach to enhancing long-term genetic gain.

Divergent selection for body weight. Selection for low body weight appears as effective as that for high body weight in Japanese quail. Nestor et al. (1982), and Darden and Marks (1988a) have demonstrated that divergent selection for four-week body weight resulted in similar response patterns in upward and downward directions. Nestor et al. (1982) reported four-week body weight regression coefficients of 3.1 and -4.2 g per

Table 27.6. Realized heritability estimates for four-week body weight in S, T, and P line quail. (From Marks, 1989).

	S line		T line		P line	
Generation	Realized[1]	Realized[2]	Realized	Realized	Realized	Realized
28-40	.19	.16±.03	.02	.06±.05	.11	.12±.04
40-50	.14	.21±.06	.05	.08±.05	.19	.20±.04
50-60	.13	.23±.07	.00	.11±.08	.16	.15±.10
60-70	.11	.01±.09	.15	.13±.08	.02	-.06±.04
28-70	.14	.12±.01	.05	.06±.01	.10	.10±.01

[1] h^2=cumulative response/cumulative selection differential.
[2] h^2=regression of population means on cumulative selection differentials.

generation following seven generations of selection in high and low body weight lines respectively. These authors observed larger selection differentials and a slightly higher realized heritability when selection was for low body weight (h^2=.37±.05 in high line and .44±.28 in low line).

Darden and Marks (1988a) investigated divergent selection for four-week body weight in Japanese quail under complete-diet and split-diet environments. Under the split diet, quail had the opportunity to self-select feed from high protein and high energy diets, whereas the complete diet consisted of a blend of the high protein and high energy diets. After 11 generations of selection, the high lines were 49 and 50 percent higher and the low lines were 47 and 45 percent lower in four-week body weight than controls (Figure 27.3). Realized heritabilities for four-week body weight were .36 and .30 for high and low lines respectively under the split diet and .52 and .47 for the high and low lines respectively under the complete diet. In this experiment, the split-diet environment could be considered less than optimum with respect to growth since the controls and selected lines were approximately ten percent smaller than comparable birds under the complete-diet environment. The selected lines under the split-diet environment exhibited larger selection differentials but only slightly smaller genetic gains to lines selected under the complete-diet environment (Figure 27.3). Therefore, the smaller heritability estimates for four-week body weight under the split-diet environment appeared to be primarily due to larger selection differentials rather than smaller genetic gains. These results are consistent with the report of Marks (1978) which showed that genetic gains were similar under a stress (T line) and nonstress (P line) environment for the initial ten generations of selection; however, heritability estimates were 30 to 40 percent larger under the nonstress environment. Therefore, it appears that initial genetic progress under optimum and less than optimum environments may be similar, although the realized heritability estimate is lower under the less than optimum environment due to the expression of greater phenotypic variation in stress environments. This greater variation could result from the involvement of a greater number of genes activated by stresses on individuals within a given population.

Physiological changes resulting from selection for growth. Evidence of major physiological changes in Japanese quail accompanying selection for increase growth is sparse. The DNA, RNA, and protein concentration of selected and nonselected lines appears similar, as does the RNA:DNA and protein:DNA ratios (Fowler et al., 1980).

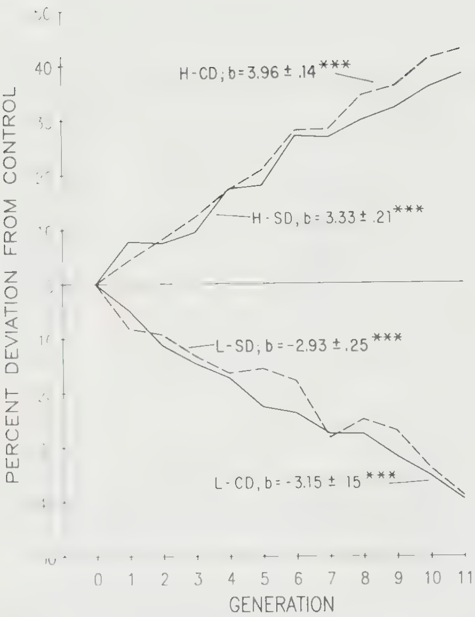

Figure 27.3. Regression of four-week body weight, as a percentage deviation from control, on generation number for high (H) and low (L) body weight lines of Japanese quail selected under complete-diet (CD) and split-diet (SD) environments.

Burke and Marks (1984) also failed to detect higher levels of growth hormone, thyroxine and triiodothyronine levels in growth selected than in nonselected lines of Japanese quail. In fact, of all physiological traits investigated, there has been little evidence of any differences between genetically diverse lines of quail. Feed intake, however, has been identified as a major factor related to genetic improvement in growth (Marks, 1981). However, feed efficiency may also be important immediately posthatch in initiating genetic diversity between selected and nonselected lines of Japanese quail (Darden and Marks 1988b).

Japanese quail selected for high four-week body weight also have more abdominal fat than nonselected quail (Darden and Marks, 1988b). Deposition of abdominal fat in quail, however, does not occur during the early rapid period of growth, and unlike chickens, males have more abdominal fat than females (Sadjadi and Becker, 1980; Darden and Marks, 1988b).

Relationship Of Body Weight To Other Quantitative Traits

There is little information available regarding the relationship between body weight and other quantitative traits in Japanese quail and essentially no information regarding genetic and phenotypic correlations. Sefton and Siegel (1974) found both phenotypic and genetic correlations between body weights at various ages to be high with the magnitude of the estimate generally decreasing as the time interval between weighings increased. These data were expected because they were part-whole correlations. The exception involved the lack of correlation of body weight at one day of age with subsequent measurements, due to the influence of egg size on hatch weight.

Macha and Becker (1976), following nine generations of divergent selection for six-week body weight, reported that the correlated response for egg production was negative in both small and large body weight females. Strong et al. (1978) observed genetic correlations between body weight at sexual maturity and egg weight of .42 and .43 respectively. The genetic correlation between body weight and 70-day egg production was essentially zero (-.04). Marks (1979) reported that following long-term selection (38 generations) for four-week body weight in two separate lines, 16-week body weight and egg weight increased whereas egg production, percent fertility, and percent hatchability declined. Marks (1980) found feed conversions of two lines (P and T) selected for high four-week body weight were superior to that of a nonselected control line following 42 generations of selection. The feed per gain ratio of the P line was 2.08 compared to 2.72 for the nonselected line indicating that selection for increased body weight also resulted in improved feed utilization. These data indicate a positive relationship between various measures of body weight to feed efficiency and between body weight and egg weight. Although sparse, the available literature on Japanese quail provides good evidence that a negative relationship exists between body weight and various reproductive parameters similar to the negative relationships observed in chickens and turkeys.

GROWTH AND MEAT PRODUCTION IN GUINEA FOWL

Information regarding quantitative growth parameters for guinea fowl is sparse. The early patterns of growth for guinea fowl, however, appear similar to the growth patterns of broiler-type chickens prior to the intense selection for increased body weight initiated in the 1940s and 1950s. Hughes and Jones (1980) reported mean four-, eight-, and 12-week body weights of 334, 676 and 1041 g when fed an adequate diet with a feed conversion (g feed per g gain) ratio of 3.24 for the 0-12 week period. Since guinea fowl have undergone little artificial selection for increased growth rate, it is very likely, based on data obtained from other avian species, that genetic responses to selection for body weight would be dramatic as in the initial selected generations in chickens and turkeys.

GROWTH AND MEAT PRODUCTION IN RING-NECKED PHEASANTS

Ring-necked pheasants are popular gamebirds that are utilized both as gourmet restaurant items as well as true gamebirds in hunting preserves. If these birds are raised for eventual release on shooting preserves, the rate of growth is not an important criterion, but rapid growth is critical if they are raised for the restaurant trade. Woodard et al. (1977) observed ten- and 20-week male body weights of 753 g and 1303 g in unselected Chinese pheasants fed a 28 percent protein diet; female body weights were 639 g and 932 g at ten and 20 weeks of age. These same authors reported that unselected Mongolian pheasants were 10-20 percent larger than Chinese pheasants, which may indicate genetic diversity between different populations of pheasants.

Kassid et al. (1981) divergently selected ring-necked pheasants for high and low 12-week body weight. Data were obtained from eight sires, 59 dams, and 1197 offspring over a period of two generations. The response in the line selected for high body weight was greater than the response in the line selected for low body weight. Average heritability estimates calculated from parent-offspring regression (omitting negative values and those greater than one) were found to range from .29 to .47, .44 to .64, .41 to .66, .20 to .77, and .33 to .63 for body weights at one day, four weeks, eight weeks, 12 weeks and 18 weeks of age respectively. Average realized heritability estimates for 12-week body weight were found to be .50 and .33 in males and females respectively in the low body weight line, and .71 and .38 in the high body weight line for males and females (Kassid, 1981). Data on 1200 ring-necked pheasants produced in three generations of selection for rapid and slow feathering were also used to estimate heritability of body weight (Hussein, 1983). Heritabilities ranged from .22 to .53 for four-week body weight, from .16 to .66 for eight-week body weight, and from .10 to .73 for 12-week body weight.

Heritability estimates for body weight in ring-necked pheasants fit nicely into the range of heritability estimates for chickens (Siegel, 1962; Kinney, 1969) and Japanese quail (Marks, 1978). The moderate to high heritability estimates for body weight in ring-necked pheasants suggest the presence of high additive genetic variance which would indicate that mass selection would be an effective tool to rapidly increase body weight in this species.

Little information is available regarding the influence of selection for increased body weight on reproductive traits in guinea fowl and ring-necked pheasants. Since these birds have not undergone as intense selection as have broiler chickens, the decline in reproductive traits would not be expected to be as great. However, there is sufficient data in broiler chickens, turkeys, and Japanese quail to suggest that long-term selection for body weight in guinea fowl and ring-necked pheasants will likely result in declines in reproductive fitness similar to those observed in chickens, turkeys and Japanese quail. An appropriate summary of these relationships can perhaps best be gleaned from a quote from Lerner (1954):

> "Attempts to shift populations too rapidly and too far from adapted mean values for specific traits, either by artificial selection or by changes in the breeding system, are counteracted by natural selection which is directed towards the maintenance of a phenotypic balance between fitness-determining characters."

CONCLUDING REMARKS

Quantitative genetic parameters of growth and meat traits in Japanese quail, guinea fowl and ring-necked pheasants demonstrate considerable variation similar to that found in chickens and turkeys (Table 27.7). Heritability estimates for body weight and growth rate range from .06 to .74 in Japanese quail and .20 to .79 in ring-necked pheasants, with mean

Table 27.7. Summary of mean heritabilities for body weight in other galliforms. Estimates calculated from populations that have undergone less than ten generation of selection.

Species	Method[1]	Trait	h^2	Range	Estimates, no.
Japanese quail	E	BW (14-58 d)	.41	.17 to .74	30
Japanese quail	R	BW (21-42 d)	.37	.06 to .68	14
Ring-necked	E	BW (1-126 d)	.50[2]	.20 to .79	15
pheasant	R	BW (84 d)	.47	.33 to .71	4

[1] Method: E=estimated from paternal and maternal half-sib correlations and by parent-offspring regression; R=realized.

[2] Estimates <0 or >1 omitted.

values of .39 and .48 respectively. The range in heritability estimates can be attributed to the method of calculation of heritabilities, selection environment, length of prior selection (history of the population), sampling and measurement errors, and numerous other variables. The range of estimates is similar to estimates for growth traits in chickens and turkeys and indicates that the genetic components of growth are likely similar in most avian species. In general, reproductive fitness appears to decline as a result of selection regardless of the avian species.

The relatively high heritability estimates (.4 to .5) for body weight and growth in other galliforms suggest that individual phenotypic selection would likely result in the same dramatic improvement in growth as has been obtained from selection in commercial broilers and turkeys.

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Chapter 28. GENETICS OF GROWTH AND MEAT PRODUCTION IN WATERFOWL

H. Pingel

GROWTH TRAITS

Waterfowl have a remarkably rapid growth during the first weeks of life. At slaughter age of 7-8 weeks in domestic ducks, nine weeks in domestic geese, and 10-11 weeks in muscovy ducks, they attain 70-80 percent of adult weight, while chicken broilers have a slaughter weight less than 40 percent of adult weight. The waterfowl species differ in growth rates and in the degree to which males grow faster than females. White Pekins typically exhibit early growth with only a slight difference (5-8 percent) in size between sexes. The early growth of muscovies is usually quite slow and they develop a marked difference in weight between sexes. At the age of 11 weeks males are about 45 percent heavier than females. In geese, males exceed females by more than ten percent up to the age of eight weeks.

Heritability. The heritability of body weight in waterfowl is comparable to that estimated for chickens and turkeys. Table 28.1 summarizes information on the heritability of body weight at slaughter age in Pekin ducks, muscovy ducks, and Italian and Kuban geese, based on half-sib and full-sib analyses. Further estimates of heritability for body weight were calculated from Pilla (1974) for muscovy ducks at the age of four, six, eight, ten and 12 weeks. The values at these different ages were 0.60, 0.52, 0.33, 0.50 and 0.26. The h^2 of body weight of Pekin ducks was 0.18 and 0.63 if estimated from regression of offspring on sires and dams respectively (Dzhubashev, 1978).

Selection. In all waterfowl species body weight has fairly high heritability. Therefore selection for body weight at market age has been an effective method of improving growth rate. Powell (1984) was able to increase the body weight of ducks at seven weeks of age from 3.25 kg to 3.80 kg by mass selection over five generations. Schneider (1988) obtained a body weight gain of 900 g in geese over five generations.

CARCASS COMPOSITION

Selection limits for growth rate have not yet been reached, but problems have arisen from consumer demand for a smaller carcass, and from the low breast meat proportion at young ages. There is some information available about the influence of age on carcass composition and particularly on breast muscle proportion.

Stadelman and Meinert (1971) concluded that the percentage of breast muscle increases more than the percentage of leg muscle (drumstick and thigh) decreases with advancing age (Table 28.2). The same tendency was observed in muscovy ducks by Pingel and Schneider (1981) (Table 28.3). As far as skin and fat are concerned there is little decrease with age.

Table 28.1. Average heritabilities of body weight at slaughter age in waterfowl.

Breed	Age, days	Sires, no.	Dams, no.	Offspring, no.		h^{2S+D}	Reference
Pekin ducks	28			511	m	0.64	Stasko (1966)
				532	f	0.43	
	42	136	650			0.34-0.56	Cerveny et al. (1986)
	49	40	120	473	m	0.28	Pingel and Jung (1979)
				680	f		
	50			665		0.65	Dujunov (1965)
	51	28	68	427		0.76	Clayton and Powell (1979)
	56	72	373	3222	m	0.31	Sochocka and Wezyk (1971)
		60	301	2399	f	0.35	
	56	84	182	1031	m	0.35	Pingel and Heimpold (1983)
		79	169	959	f	0.43	
	56	381	940	3242	m	0.43	Kain (1988)
		365	937	3340	f	0.41	
	1-56	12	72	1160		0.23-0.89	Kosba et al. (1981)
Muscovy ducks	70	160	626	4453	m	0.43	Ricard et al. (1983)
		160	626	4452	f	0.48	
	74	344		3244	m	0.53	Trettner (1984)
	67	360		4765	f	0.49	
Italian geese	42	45	184	1107		0.34	Stasko and Masar (1968)
	84	45	184	1107		0.45	
	56	230	575		m	0.57-0.42	Wezyk and Sochocka (1976)
					f	0.69-0.42	
Kuban geese	56	50	250	1331		0.30	Mazanowski (1986)
	84	50	250	1331		0.32	

This is a surprising fact since older individuals tend to be leaner than younger ones. Romboli and Fedeli-Avanzi (1979) have found in muscovies that breast muscle tends to continue its growth when body weight has stabilized. At the age of 30, 60, 71 and 78 days the percentage of breast muscle to total muscle was 9.3, 22.7, 28.4 and 36.4 in males, and 8.0, 26.0, 35.0 and 40.0 in females respectively. Investigations reported by Bielinski (1979) on the optimal age for slaughter of geese indicated that breast muscle in relation to weight of eviscerated carcass increased from 12.7 to 17.3 percent between eight and 12 weeks of age.

Waterfowl from the beginning have a high proportion of skin and subcutaneous fat for protection against cold water. Because of the much higher surface area to total weight at

Table 28.2. The effect of age on tissue distribution in the carcass of Pekin ducks. (Adapted from Stadelman and Meinert, 1977).

	Percentage of carcass without neck and giblets		
Age, days	Breast muscle	Leg muscle	Skin and fat
28	4.8	18.0	41.6
35	7.0	16.7	41.0
42	8.4	14.7	40.4
49	12.0	14.0	38.1
56	14.4	13.4	37.1
63	15.9	12.3	37.5

Table 28.3. The effect of age on tissue distribution in the carcass of muscovy ducks. (Adapted from Pingel and Schneider, 1981).

	Males				Females			
Age, wks	Body weight, g	Breast muscle, %	Leg muscle, %	Skin +fat, %	Body weight, g	Breast muscle, %	Leg muscle, %	Skin +fat, %
7	1789	5.4	21.0	27.9	1431	9.0	18.6	25.6
8	2050	8.3	21.4	25.7	1654	13.0	18.7	25.4
9	2537	9.5	19.7	23.2	1674	14.3	17.0	21.7
10	2678	14.7	19.5	19.4	1739	20.0	16.9	19.2
11	3070	16.8	17.6	20.0	1759	23.4	15.1	16.0

young ages, the percentage of skin and adhering fat actually decreases as ducks approach slaughter age just before the juvenile molt. Pingel (1986) has shown that the ratio of muscle to skin including subcutaneous fat in waterfowl changes in favor of muscle with increasing age.

According to these observations, it would appear that a slight increase in age at processing could be advantageous. The percentage of carcass in the breast muscle may be increased, and the percentage of skin and fat may be decreased, to give a meatier product with lower fat content and likely with greater consumer acceptability. The intensive growth of breast muscle during the last two weeks before the beginning of the juvenile molt is related to the development of wing feathers, although flying ability is no longer characteristic of most breeds, because of considerable increase in body weight and simultaneously reduced growth of the distal part of the wings.

BREAST MUSCLING

An objective in successful waterfowl breeding is to increase the proportion of breast muscle. Then it is possible to reduce the age at slaughter with an associated improvement in feed efficiency. There are two possible ways of improving the meat proportion of waterfowl by breeding: firstly by crossing commercial breeds with breeds that can fly and have a high percentage of breast muscle, and secondly by selection.

Rudolph (1965) and Luehmann (1967) have crossed Call ducks with Pekins, Indian Runners, and Khaki Campbells to produce small broiler ducks with high breast meat yield. The Call duck plays a considerable role because of its body conformation which is similar to that of mallards. A comparison between carcass quality of F_1 (Call x White Pekin) and that of White Pekin ducks has indicated that the hybrids may be considered valuable for the breeding of a broiler duck. Majna et al. (1971) compared a strain bred from Pekin, Campbell and mallard with Pekin ducks. At the age of 56 days the body weights were 1.65 kg and 2.51 kg and feed conversions were 3.52 and 2.96 kg respectively. Because of early reduction in growth rate there was an unfavorable influence on feed conversion in the mallard crossbreds. Therefore such crossbreds have to be improved by selection for growth rate and feed conversion. There are no reports on crossing of flying geese with domesticated geese, but some local goose breeds should be tested if it can be shown that they produce more breast muscle.

Intergeneric hybrids resulting from mating between muscovy drakes and domestic ducks have been used increasingly for fat liver production. These 'mulards' have reduced sex dimorphism compared to muscovy ducks. More attention should be paid to using these sterile hybrids, for instance from the crossing of muscovy drakes and Pekin ducks, to combine the high proportion of muscle of the first with the high reproductive rate of the second.

In a study by the author, ten males and ten females from each of muscovy, Pekin, and

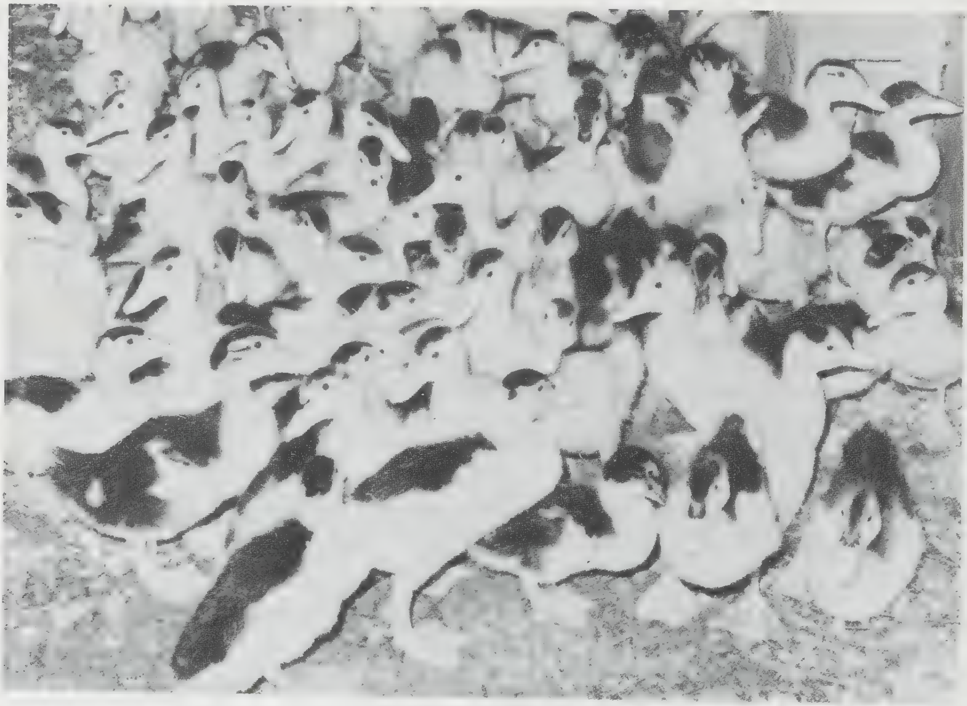

Figure 28.1. Young mulards, hybrids of muscovy drake x Pekin duck.

mulard were dressed at six weeks of age and at the usual slaughter age as determined by the end of the rapid growth period. The proportion of grill-ready carcass was influenced by age in both species and their crossbreds (Table 28.4). Hybrids almost equalled purebred muscovy ducks regarding proportion of muscle to carcass. They exceeded markedly the muscle proportion of Pekin ducks (Figure 28.2). At six weeks of age all birds had a low proportion of breast muscle. An earlier slaughter age results in carcasses with low meat yield. But mulards reached slaughter age two weeks earlier than muscovy drakes. The decrease in skin and subcutaneous fat with age in muscovy ducks and mulards indicates that the meat:fat ratio in the carcass will be altered in favor of meat.

It has been shown that in contrast to body growth, the breast muscle tends to continue its growth up to a greater age. This is the reason for the low relationship between the body weight and breast muscle expressed as a percentage of the carcass (Table 28.5). Kain (1988) has found a genetic correlation between body weight and percentage of breast and leg muscle of r_g=-0.21. Clayton and Powell (1979) estimated a genetic correlation of r_g=-0.38 between body weight and breast muscle proportion. Selection to increase body weight might depress the meatiness of a carcass. Therefore the percentage of breast muscle should be used as an additional selection criterion.

Measurement. Breeding for higher breast meat yield can be based on estimated breast muscling in live birds themselves or on direct measurements in their relatives, for instance from dressing and dissecting progeny groups. Visual inspection and handling of live waterfowl is ineffective as an indicator of carcass composition, especially of breast muscle percentage, because of dense feathering on a cylindrical body. Pingel et al. (1969) used a probe to estimate breast layer thickness (Figure 28.3). They found positive correlations between this measurement and breast muscle percentage of carcasses. Similar correlations were found in muscovy ducks and geese. They made the important observation that there was no correlation between breast layer thickness and percentage of skin and subcutaneous

Table 28.4. Carcass composition in ducks slaughtered at different ages.

				Percentage of carcass			
Species	Age, wks	Live weight, g	Dressing percentage	Breast muscle	Whole muscle	Skin + fat	Bones
Muscovy ducks							
males	6	1564	55.6	3.7	45.5	29.9	22.6
	11	3126	65.2	16.0	59.3	20.2	20.5
females	6	1045	57.7	4.1	49.9	29.9	20.2
	9	1932	63.3	14.6	55.6	23.4	21.0
Pekin ducks							
males	6	1859	60.6	6.3	45.7	30.6	23.6
	8	2707	63.9	11.6	45.0	36.1	18.9
females	6	1874	62.8	7.1	45.7	31.9	22.4
	8	2610	64.3	12.9	45.2	35.4	19.3
Mulards							
males	6	2235	63.0	6.9	48.5	29.0	22.5
	9	3234	67.7	16.3	55.3	23.6	21.2
females	6	2258	63.4	6.3	45.4	30.7	23.9
	9	2993	68.3	15.3	54.6	26.0	19.4

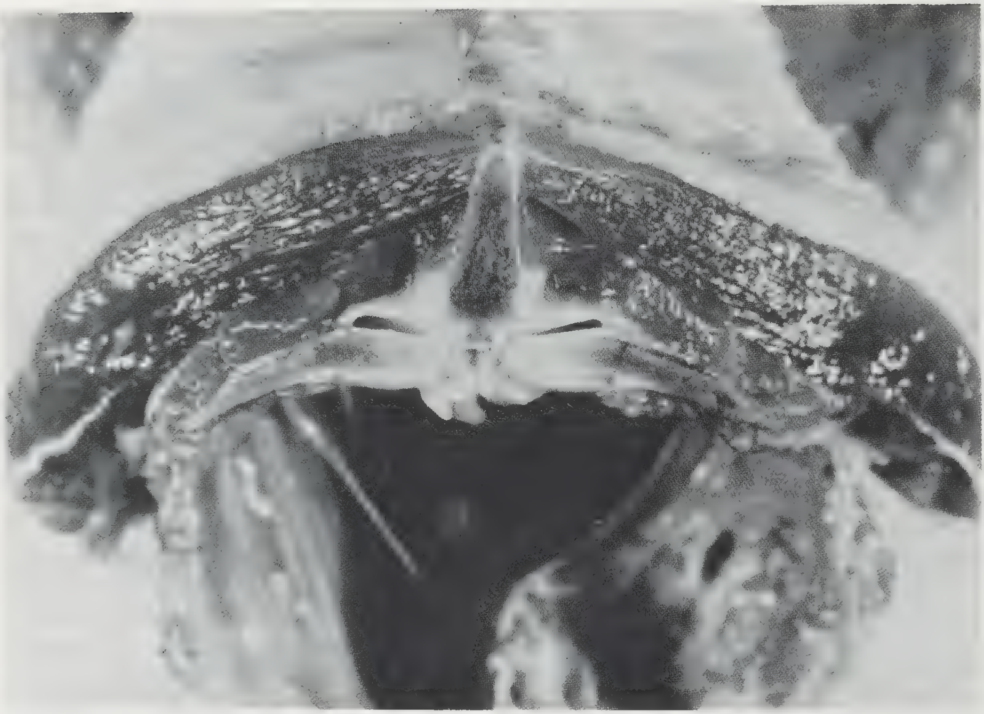

Figure 28.2. Cross-section of a mulard carcass.

fat. Janiszewska et al. (1982) used the needle probe to measure thickness of the breast layer of ducks at three and seven weeks of age. Macroscopic analysis of breast muscles showed no changes in color and structure of the probed muscles. Bochno et al. (1978) and Borsting (1981) acknowledged that thickness measurements of the breast layer can be taken as objective and reliable indexes of meatiness in live birds. This underlines their applicability to selection purposes in waterfowl.

Table 28.5. Phenotypic correlations of body weight and thickness of breast layer with percentage of breast muscle and skin.

		Percentage of breast muscle	Percentage of breast skin
Body weight	Pekin ducks	0.23	0.19
	Muscovy ducks	0.08	0.14
	Geese	0.10	0.14
Thickness of breast layer	Pekin ducks	0.59	0.06
	Muscovy ducks	0.41	-0.02
	Geese	0.55	0.05

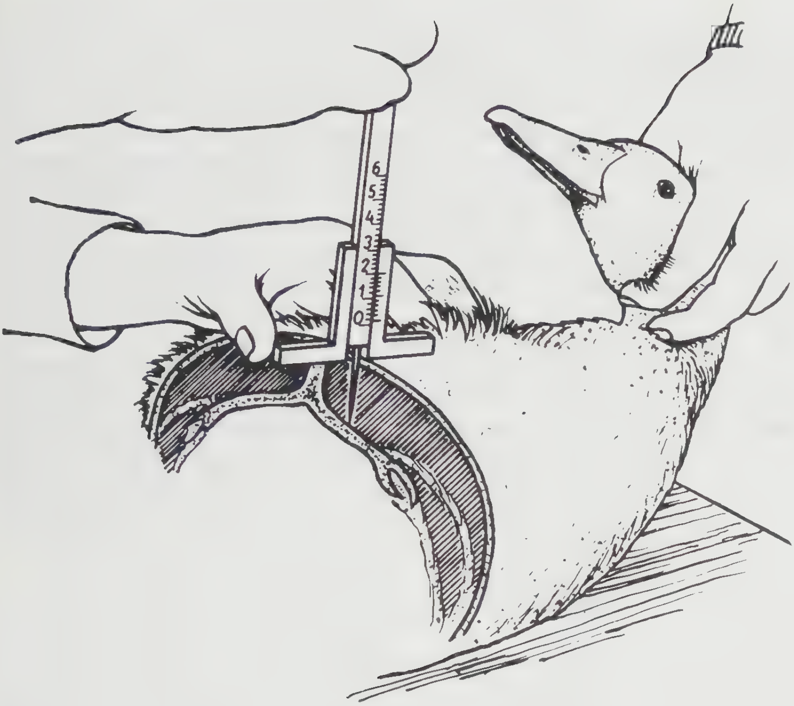

Figure 28.3. A probe for measurement of breast layer thickness in ducks.

Selection. In a selection study with Pekin ducks, Pingel and Jung (1979) determined a realized heritability of 0.51 for breast layer thickness. Pingel and Heimpold (1983) selected over seven generations for high body weight and high breast layer thickness. The selection procedure was based on independent culling limits. The cumulative selection difference for body weight was 1288 g and for breast layer thickness 9.1 mm. They were adjusted for the number of offspring. The realized heritabilities for the two traits were 0.39 and 0.32, confirming the estimated values. At eight weeks of age, body weight was improved by 18.2 percent, breast layer thickness by 17.2 percent, and breast muscle percentage as a correlated trait by 9.4 percent (Table 28.6). Despite a 500 g increase in body weight the carcass became meatier. The change in leg muscling indicates a slightly negative correlation between breast and leg muscle proportions. Kain (1988) calculated a negative genetic correlation of r_g=-0.10 between these traits. It can be explained by the opposite pattern of breast and leg muscle development with increasing age.

Borsting (1981) has also used the needle method in live ducks for determining thickness of the breast layer. The breast muscle percentage of the duck strain which had been intensively selected for high breast layer thickness showed a considerable increase. The difference between the selected and nonselected strain increased from 0.7 to 2.7 percent over three years (Table 28.7).

Direct selection for breast muscle proportion involves the slaughter and dissection of sibs or offspring. Heritability studies of breast muscle proportion have indicated that this trait should respond to selection. The estimated h^2 values were 0.62 (Stasko, 1965), 0.65 (Clayton and Powell, 1979) and 0.38 (Kain, 1988). Powell (1984) found that selection for proportion of breast muscle based on progeny test information does not necessarily result in improved feed conversion. Table 28.8 shows that with increasing breast muscle proportion, the feed conversion actually deteriorated.

Stasko (1980) selected for breast meat proportion based on slaughter analysis of two brothers and two sisters. The expected increase in breast meat proportion was realized. However the proportion of leg parts decreased slightly. Accordingly subsequent selection included both breast and leg meat. After three generations an improvement of about 2.0 percent in breast meat proportion was recorded, but there was only 0.5 percent improvement in the proportion of legs. The response of leg muscle percentage to selection is lower than that of breast muscle percentage because of the decrease in genetic variation for leg muscle proportion with advancing age.

Table 28.6. Body weight, breast layer thickness, and percentage of breast muscle in carcass after seven generations in a duck breeding experiment. (Adapted from Pingel and Heimpold, 1983).

	Selected line	Control line	Difference, %
n	675	689	
Live weight, 8 wks, g	3287	2782	18.2
Breast layer thickness, mm	19.8	16.9	17.2
n	463	562	
Breast muscle in carcass, %	13.0	11.9	9.4
Breast skin in carcass, %	6.0	6.3	-5.0
Whole leg in carcass, %	19.8	20.1	-1.5

Table 28.7. Selection for thickness of breast layer in ducks. (Adapted from Borsting, 1981).

	Nonselected strain			Selected strain		
Year	Live weight, g	Thickness of breast meat, mm	Percentage of breast meat	Live weight, g	Thickness of breast meat, mm	Percentage of breast meat
1975	2980	172	14.1	2480	16.2	14.8
1978	2950	174	13.6	2480	17.8	16.3

Table 28.8. The results of selecting for improved proportion of breast muscle over five generations. (Adapted from Powell, 1984).

	Live weight, g	Feed conv, kg	Breast muscle weight, g	Breast muscle proportion, %
Control	2782	2.586	189	12.4
Selected	2849	2.715	268	15.7

Effects on meat quality. Arising from improvements through selection for breast muscle content, meat quality has been compared between ducks with larger and smaller breast muscle percentages. The comparison between ducks with high and low breast muscle content has shown that pH values, color, and cooking losses of breast meat have not been affected (Table 28.9). Heritability estimates of meat quality properties were low. These data suggest the conclusion that environmental factors have a greater effect on meat properties than does genetics.

Effects on leg disorders. The selection for rapid growth has some other negative consequences. Wise and Nott (1975) observed tibial dyschondroplasia in nine strains of ducks. The incidence was higher in the heavy strains than in the light strains. But it was concluded that tibial dyschondroplasia seldom caused economic losses. The incidence can be changed if waterfowl are kept on wire floors.

Pingel et al. (1987) found a high incidence of leg disorders in heavy Pekin ducks kept in single cages on wire floors. Males appeared to be twice as sensitive as females. Individual selection against leg disorders reduced them markedly after two generations. From this preliminary test it would appear that twisted legs are the main category of leg disorders.

FEED CONVERSION

Selection for rapid growth rate means that ducklings and goslings reach slaughter weight at younger ages and thus feed conversion will be improved by the reduction of maintenance requirement. A reduction of one day in reaching a certain weight reduces feed consumption of Pekin ducks by 100 to 120 g. This means an improvement in feed conversion by 0.04 kg or 1.3 percent. However the marketing of ducks at younger ages has brought problems of lowered breast muscle percentage and higher subcutaneous fat deposition. Therefore selection for better feed conversion should be introduced. Such practices have already been used by Pingel et al. (1984) and Powell (1984). The latter author selected directly for better feed conversion in three lines. He found a considerable response in feed conversion as well as correlated reductions in the proportion of skin and fat

Table 28.9. Meat quality of ducks with high and low breast proportion. (Adapted from Pingel and Birla, 1982).

	n	pH value	Color remission	Cooking losses, %
Strain A				
Breast meat proportion over 14%	45	5.84	8.6	23.3
Breast meat proportion below 11%	50	5.88	9.7	25.5
Strain B				
Breast meat proportion over 14%	22	5.93	9.7	23.6
Breast meat proportion below 11%	22	5.94	9.9	25.4

after nine generations (see Chapter 40 Table 40.2).

In an experiment conducted by the author, using a synthetic population created by crossing White Call and Pekin ducks, selection was conducted for low feed conversion (LF) versus high feed conversion (HF). The initial population was continued as a control without selection and with random mating. All three lines consisted of 20 pens each with one drake and two ducks. The calculation of feed conversion was based on feed consumption and body weight gain of individuals from 3-7 weeks of age. The results (Table 28.10) show quite clearly that direct selection for feed conversion was successful after eight generations. In the LF line the cumulative selection difference was -2907 g and the selection success of both sexes was -926 g. In the HF line selection difference and selection response were lower at +2299 g and +386 g, because it is difficult to find ducks with high body weight and low feed conversion. The realized h^2 value for feed conversion was 0.32 for ducks of the LF line. In the HF line the corresponding h^2 value was 0.17.

This two-way selection experiment was conducted primarily to study the effect of feed conversion on carcass composition and its nutrient content. There was an opposite response in muscles and skin. In the LF line the birds showed a significantly higher proportion of leg muscle than that of the HF line. An entirely different response was shown for skin proportion. Since large amounts of fat are deposited especially in the subcutaneous tissue of ducks, this finding may be reflected in a lower carcass fat content. Carcasses of the LF line generally showed a marked reduction in fat content. The results obtained from studies on composition and nutrient content of dressed carcasses are in good agreement. They allow the conclusion that selection for low feed conversion results in an increase in muscle proportions, but causes a considerable decrease in skin and fat content. The reduction of skin and depot fat proportion may be considered to be a cause of improved feed conversion although it may not be regarded as the only cause.

Theoretical examination of the problem makes it clear (Powell, 1985) that individuals with superior feed conversion should have a lower proportion of fat, since protein in the

Table 28.10. Effect of divergent selection for feed conversion ratio in ducks on carcass composition and fat content after eight generations. (Adapted from Klemm and Pingel, 1988).

	Males			Females		
	LF	HF	Control	LF	HF	Control
n	113	149	138	80	136	150
FCR, 28-48 d, kg	3.133	4.349	3.763	3.445	4.820	4.266
Live wt, 48 d, g	2733	2263	2336	2608	2042	2142
Proportions of carcass						
n	50	39	41	25	25	31
Breast muscle, %	12.0	13.1	12.5	13.6	13.0	13.6
Leg muscles + bones, %	15.0	12.9	13.3	13.8	13.1	13.0
Breast + leg skin, %	11.5	15.0	13.1	10.7	14.0	13.2
Cooking loss, %						
Breast muscle	21.8	22.9	22.9	24.9	24.3	23.7
Whole leg	41.5	50.8	46.3	41.9	53.1	50.1
Fat content	21.7	30.1	23.5			

carcass is associated with approximately 70 percent water in muscle tissue and fat with only about ten percent water in fatty tissues. As protein and fat are deposited with almost the same energy cost, the deposition of muscle tissue should be approximately three times as efficient as the deposition of fat. This is the reason that selecting for improved feed conversion leads to a correlated reduction in fat content.

Improvements in feed conversion cause differences in metabolism and physiological parameters. Mueller (1986) was able to show different enzymatic activities in ducks of the LF and HF lines. The activity of alkaline phosphatase was especially higher in the LF line, but more research is needed to find the relationship between feed conversion and enzymatic activities. Genetic improvement of feed efficiency has to be explained by further analysis of physiological traits, as well as behaviors such as locomotor activity.

Waterfowl, as grass consuming birds play an important role in utilizing areas which could not otherwise be used. Therefore the genetic variation in utilization of local resources (specific feedstuffs) deserves more attention. There is no evidence that the efficiency of grass converting by waterfowl is influenced by additive genetic factors.

FAT CONTENT

Veremejenko (1987) has applied family selection for lower fat content in ducks. After three generations the fat content was reduced from 29.4 to 24.3 percent. Unfortunately it was not compared to feed conversion.

A minimum of subcutaneous fat is necessary, because the desirability of roasted waterfowl is greatly influenced by the crispness of the skin. Of special interest is the fact that waterfowl fat has a favorable fatty acid composition (Pereira and Stadelman, 1976). The percentages of major fatty acids in duck lipids are: oleic 52.8, palmitic 21.4, linoleic 14.3, stearic 5.6, and palmitoleic 4.8. The high unsaturation level for duck lipids of about 73 percent and the polyunsaturated:saturated fatty acid ratio of 0.54 is characteristic of duck fat. A similar fatty acid composition was found in depot fat of geese by Friend et al. (1983).

FATTY LIVER

In some countries production of goose fatty liver has been developed. This has been accompanied by breeding of populations with high liver weight. Appropriate breeds for fat liver production are Landaise and Slovak geese and especially their hybrids (Stasko, 1976). These breeds have a lower threshold of overfattening by force feeding. The lower fattening threshold is a manifestation of the ability to produce a large liver without overfattening of the body.

Heritability of liver weight estimated by Stasko and Masar (1968) in Italian geese was 0.42 to 0.48, which agrees with heritability for body weight. Breeders endeavor to find methods to estimate liver size without slaughter. Attention has been paid to investigation of biochemical changes in liver and to their use as markers. Czuka and Koci (1976) observed contradictory correlations between liver size and cholesterol activity, amylase activity, bilirubin content, and serum refraction indicating that these markers cannot be generally used for testing liver size. More promising markers for liver size are serum transaminase GOT and GPT.

Rouvier et al. (1982a) proposed a selection index based on collaterals to increase the fat liver weight of Landaise geese. Rouvier et al. (1982b) increased the mean fat liver weights for two strains of Landaise geese to 773 g and 732 g respectively. Phenotypic correlations showed that average daily weight gains during force feeding can be used to predict fat liver weights.

In recent years intergeneric hybrids resulting from mating muscovy drakes to domestic ducks have had more utilization for fat liver production. Veremejenko (1987) produced livers of 160 to 450 g by crossing muscovy drakes with White Ukrainian ducks. Babile et al. (1988) have found that force-fed mulards had higher body weight gain during cramming than muscovy ducks. Their fatty liver weight was higher by 98 g but the melting loss during sterilization at 105° C was lower by ten percent.

FEATHERS

Waterfowl are characterized by dense covering of skin and feathers. The relative weight of feathers in geese is considerably higher than that in chickens. The dynamics of juvenile feathering are connected with body growth. The highest correlation between feathering intensity and body weight in ducks was found at the age of 28 days (Majna et al., 1971).

The growth of young feathers in geese lasts 49 days, beginning from the third week of life. The first plucking should be done between 70 and 77 days of age, when the feathers have attained full maturity. The quantity of down feathers in three successive pluckings was investigated by Bielinski (1979). In growing geese the amount of down was 43.9 g, and in adult geese it was 54.5 g (Table 28.11). The effect of selection for an increased amount of down per unit of skin surface should be investigated.

Table 28.11. Quantity of down and feathers (g) from three pluckings. (Adapted from Bielinski, 1979).

	Growing geese			Adult geese		
Successive plucking	Feathers	Down	Total	Feathers	Down	Total
1	40.3	4.7	45.0	71.2	18.0	89.2
2	70.7	16.9	87.6	57.6	15.4	73.0
3	87.6	22.3	109.9	88.9	21.1	110.0
Total	198.6	43.9	242.5	217.7	54.5	272.2

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Chapter 29. GENETICS OF EGG PRODUCTION IN CHICKENS

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The egg production of a chicken is a result of many genes acting on a large number of biochemical processes, which in turn control a range of anatomical and physiological traits. With appropriate environmental conditions (nutrition, light, ambient temperature, water, freedom from disease, etc.), the many genes controlling all the processes associated with egg production can act to allow the chicken to express fully its genetic potential.

How egg production is measured determines what part of the genome of the bird is being considered. If egg production is measured from age at first egg, the trait probably excludes many genes affecting sexual maturity. If only early egg production (part-record) is considered then genes responsible for persistency, that do not act on early production, are not included. Egg production measured only in a second laying cycle does not consider genes influencing first laying cycle egg production alone, and so on. Thus, the definition of egg production and how it was measured in the studies reviewed are crucial to understanding the results.

Scientific investigations into the genetics of egg production began around 1900 (see the reviews in Hutt, 1949, and in Lerner, 1950, 1958 for detail). Much of the early work on egg production was Mendelian in the classical sense and focused on classification of absolute genetic effects. At the Maine Agricultural Experiment Station, Gowell (see Pearl and Surface, 1909) tested the efficacy of mass selection to improve egg production (a notable failure in this case). Gowell's successor, Pearl (1915), was among the first to publish the low correlation between the genotype and phenotype, while still trying to fit egg production into a Mendelian pattern. Dryden (1921) was one of the first workers to demonstrate substantial improvement in egg production due to selection, selecting females on their own individual records and males on the performance of their dams. Goodale (Goodale and Sanborn, 1922), while continuing to follow the Mendelian theme, described separate biologically related factors of egg production, such as sexual maturity, rate of lay, and persistency. Hurst (1925) subdivided the egg production record into component part-records which were each fitted to a Mendelian frequency pattern. Munro (1936) at the Central Experimental Farm in Ottawa proposed a polygenic inheritance for egg production and was the first to estimate genetic variation as a proportion of phenotypic variance.

This chapter will introduce the reader to definitions of populations and traits associated with measures of egg production, to some important environmental and endogenous influences on egg production, to mathematical descriptions of the egg production curve, to heritabilities of egg production traits and their genetic relationships with other important traits in chickens, and to the literature on selection for improved egg production. Criteria and methods of multiple trait selection as they relate to selection for improved egg production and related economic traits in laying hens will be discussed, but not in the detail required for application. Finally, some implications of selection and experimental results with regard to selection for high egg production will be discussed.

MEASURES OF EGG PRODUCTION

Populations

Distinct populations that form the basis of an egg production measurement can be defined. Two of the most common are the hen-housed and survivor populations.

The *hen-housed population* consists of all hens placed in the laying house (or housed) at the end of the rearing period. If a hen dies after being housed, she is still considered a part of the population for the purposes of measuring egg production. The population is usually housed before the hens are laying a significant number of eggs.

The *survivor population* consists only of hens surviving to the end of test, usually after about a year (365 days) in the laying house. However, shorter test periods are often used. The shorter the period, the less meaning there is to the term survivor. To eliminate the effects of morbidity, further criteria can be added, such as a minimum rate of lay, to eliminate records of hens suffering from subclinical diseases. How these criteria are applied can subtly affect the defined population. For example, if a minimum rate of lay after a hen's first egg is a criterion, applying this exclusion across the complete laying period will define a different population than compliance with this restriction in each of several subperiods (e.g. each third of the laying year). In the former case, many hens affected by subclinical disease contracted after the onset of lay will not be excluded from the population even if they go out of lay for a substantial part of the laying period.

There are other less well defined populations, for instance all birds with a valid observation for a particular trait. This is not one uniform population, but a series of populations that may vary slightly for each character. This population is often implicit, especially in correlation data where the population consists of that subset of individuals with a valid observation for both traits (or each of a group of traits).

Egg Production Traits

Egg production can be measured as a number of eggs or as rate of egg production. *Hen-housed egg production* (hen-housed egg number) is the number of eggs that a hen lays after placement in the laying house. For hen-housed egg production, zero eggs is a valid observation for an individual hen. Hens that die subsequent to placement in the laying house and hens that never lay are part of the hen-housed population, and they contribute to hen-housed egg production although their records are near or at zero. Hen-housed egg production is a combination of three component traits: age at first egg (AFE), rate of egg production from the start of egg production, and viability (including morbidity or any other factor causing production to cease). When dealing with this trait, it must be understood that any of these three traits can profoundly affect hen-housed egg production.

Hen-day rate of egg production is the number of eggs laid divided by the number of days a hen was alive in the period (sometimes called hen days). Calculations may commence at housing, at some other arbitrary age, or at the onset of egg production for each hen. If the hen days are calculated starting from AFE of individual hens, the effects of sexual maturity (AFE) and viability on the hen-day rate of egg production record are reduced. Hen-day rate of egg production is not free from morbidity.

Rate of egg production from housing or hen-housed rate of egg production is simply another way of expressing hen-housed egg production. Hen-housed egg number or rate of egg production in the past has been referred to as the *production index* since it is a crude index of the three component traits (above).

INFLUENCES ON EGG PRODUCTION

Age

Age influences egg production within the first laying cycle and over subsequent laying cycles. In each laying cycle, egg production (per hen housed or per live hen) quickly rises to a peak and decays slowly thereafter to the end of the cycle (see Fairfull, 1982; Gowe and Fairfull, 1982a), usually terminating with a natural or induced molt. In successive cycles of egg production, the peak egg production of a flock is usually lower and the rate of decay of egg production is more rapid.

The quick sigmoid rise to peak egg production arises mainly due to differences in age at first egg (sexual maturity) of individual hens in the population. When egg production records are synchronized for sexual maturity, the 'hook' at the start of the production record largely disappears (see Gavora et al., 1982; McMillan et al., 1986). The decline of the egg production curve results from a reduced rate of lay and, in the case of hen-housed egg production, from mortality.

In large studies, with populations well structured for variance component estimation, changes in the heritability of egg production traits with increasing age are customarily small. The heritability of hen-housed egg production usually decreases (Van Vleck and Doolittle, 1964; Clayton and Robertson, 1966; Gowe, 1977; Liljedahl et al., 1984; Weyde and Liljedahl, 1984; Gowe and Fairfull, 1986), while that of survivor egg production or rate of lay usually increases (von Krosigk, 1975; Emsley et al., 1977; Flock, 1977; Gowe, 1977; Gowe and Fairfull, 1986) with age in the first laying cycle. Ordinarily, these changes are on the order of 1-5 percent.

Changes in the variation of egg production traits with changes in the age of the bird are much larger than would be predicted from heritabilities. Additive genetic variation, as estimated from the sire variance component, increases during the first laying cycle by about 1.5-2 times for egg production traits (Liljedahl et al., 1984; Liljedahl and Engstrom, 1986). Increases of a similar order occur with respect to nonadditive genetic variation. The relatively small changes in heritability occur because environmental variation, and thus phenotypic variation, experiences relatively large increases with increased age. Liljedahl et al. (1984) hypothesized that this increased genetic variation can be attributed to the expression of an increased number of genes determining egg production at later ages. If true, this would be a sound reason for caution in using too short a part-record of egg production as a basis for selection.

In summary, within egg production cycles, egg production declines with increasing age while its variation increases. Over successive egg production cycles, egg production peaks at a lower rate and the decline of egg production is more rapid.

Disease

Disease affects egg production through mortality and morbidity (subclinical and clinical disease, inadequate nutrition, toxic elements, etc.). Mortality reduces the number of layers available to lay eggs, and morbidity reduces the laying ability of affected hens. The effects of morbidity and mortality on egg production records depend upon the age of the hens when affected. Also, mortality and morbidity can significantly affect genetic and environmental variation, heritabilities, and the accuracy of selection. These and related issues are discussed in more detail in Chapter 33.

Single Gene Effects

Single genes can exert an effect on several traits of the phenotype due to pleiotropy or to linkage. Unless linkage is very close, effects due to linkage tend to disappear over time as the linkage breaks up because of crossing over at meiosis, whereas pleiotropic effects are permanent. A number of genes known to control other traits also affect egg production (see Chapter 20). A few well known examples of these effects on egg production will be discussed briefly here.

Genes that reduce body size and consequently maintenance requirements in parent stocks of broilers and in grandparent, parent and commercial layer stocks have been tested both experimentally and commercially. Hutt (1959) reported a sex-linked dwarfing gene (*dw*) of New Hampshire origin that reduced egg numbers and rate of egg production three months postpeak in New Hampshires and in Leghorn crosses. Bell et al. (1983) confirmed this finding in a Leghorn genetic background with respect to hen-day rate of egg production in the full year at two housing densities and four protein levels. Telloni et al. (1973) found that hens carrying the *dw* allele laid fewer eggs than dw^B hens, even when hens of the same body size were compared. Coquerelle and Mérat (1979) compared dw^B from the dwarf Ardennais with Dw^+ from a Rhode Island Red genetic background; in addition to reduced body size, hens carrying the dw^B allele had significantly lower egg production. The use of the *dw* gene to reduce body size of the parent female in broiler hatching egg production by commercial breeders has been reasonably successful. This no doubt can be partly attributed to selection for modifying genes that are positive for egg production, and to the use of strain or breed crosses for the female parent so that heterosis at least partially overcomes the depressing effect of the gene. In recent tests at Ploufragan, dwarf broiler females layed as well as normal broiler females (Quemeneur et al., 1986, 1988). However, these results with commercial broilers suggest the possibility that the lower egg production of dwarf chickens may be due to close linkage of the dwarfing gene and genes determining egg production on the sex chromosome. Refer to Chapter 20 for a full discussion of effects associated with genes causing dwarfism.

The major histocompatability complex (MHC), also known as the B blood-type locus (*Ea-B*), has been studied intensively in chickens mainly because of its importance with regard to disease resistance. However, the MHC has been found to influence other characters as well. In two White Leghorn lines each carrying four *Ea-B* alleles, two of which were common to both lines, Nordskog et al. (1973) reported that one allele had a consistently negative effect on egg production, and in one of the two genetic backgrounds, two alleles were associated with higher egg production. Gebriel et al. (1984) using two White Leghorn lines found that, of five *Ea-B* alleles present in both lines, two alleles were associated with higher than average egg production and one with lower egg production. In view of the inconsistent results across lines, linkage or exposure to a pathogen may be involved in the association of egg production and *Ea-B* alleles. Refer to Chapters 20, 22 and 33 for further detailed discussion and interpretation.

The native South American breed, the Araucana, is known for its blue eggshells. The blue eggshell gene (*O*) is autosomal dominant. Sadjadi et al. (1983) found that in a White Leghorn genetic background, hens laying blue-shelled (*Oo*) eggs laid at a significantly lower rate than relatives laying white-shelled (*oo*) eggs.

The slow feathering gene (*K*) has sometimes been found to be associated with reduced egg production and this was assumed to be due to pleiotropic effects (see Chapter 20 for review). Recent research suggests that morbidity and mortality may be the indirect cause of the lower production. The evidence indicates that the negative effects of the *K* gene are likely due to close linkage between the endogenous viral gene *ev-21* and the *K* allele (Bacon et al., 1988). Birds carrying the *K* gene are immunotolerant to both endogenous and exogenous leukosis viruses. The exogenous viruses have been shown to reduce production in clinically normal hens as well as increasing mortality from other causes. This subject is discussed further in Chapter 33.

Lighting

The oviposition interval is hormonally controlled, mediated by a circadian rhythm resulting at least in part from a 24-hour light-dark cycle (24-hour day). The ovulation interval is normally slightly longer than the oviposition interval. The hen starts a clutch by laying early in the day, and usually lays at a slightly later time each day, until she skips a day and lays early in the day again (starts a new clutch). If the hen did not lay later each day, there would still be a limit of one egg per day or 365 eggs per calendar year on her potential maximum egg production. This limit is a real problem as progress toward a goal would be expected to decrease as the goal is approached. This is due to the lack of useful variation to sustain progress and the resultant reduction in the intensity of selection. Hen-housed egg production of commercial stocks frequently averages 300 or more eggs per year for well managed flocks in good environments.

There appear to be two approaches to continued progress in egg production as it nears this biological limit: extending the normal laying cycle beyond a year (improving persistency) or using hens through two or three cycles of production, and breaking the one egg per day limit. Extending the length of the laying cycle or using multiple cycles poses problems such as extending the generation interval. However, known methods of breeding and management could be used, and changes in conventional breeding plans and environments would not be needed. Therefore, this approach will not be discussed further here.

To break the 24-hour oviposition limit requires modification of the environment with respect to lighting. Thus, it would be necessary to modify the length of the circadian rhythm, assuming that it is at least partially influenced by the 24-hour light-dark cycle. Also, this assumes that the ovulation interval can be modified (i.e. that genetic variation exists).

There are four possible lighting options that could be used to modify the circadian rhythm: ahemeral lighting schemes, continuous light, continuous darkness, and intermittent light (or extreme ahemeral schemes). *Ahemeral lighting schemes* refer to light-dark cycles of less than or more than 24 hours. To circumvent a limit of one egg in a 24-hour day, the ahemeral schemes of interest are those of less than 24 hours. Variation in the ovulation interval exists (Abplanalp, 1966; Biellier, 1985). Studies using ahemeral lighting schemes of more than 24 hours have shown that it is possible to increase the ovulation interval (Morris, 1973; Biellier, 1985). Also, McClung et al. (1976) selected for reduced oviposition time under 24-hour light-dark cycles and obtained a significant response.

Thus, it is logical to assume that the ovulation interval can be decreased. The assumption is that there is an underlying genetic distribution which cannot be fully expressed (the distribution is truncated) due to the limit of the 24-hour light-dark cycle (see Biellier, 1985), but that this variation could be expressed in a nonlimiting environment (i.e. a shorter day, or continuous light, etc.).

Abplanalp (1966) studied the effects of continuous darkness and intermittent light of one minute per hour on chickens. Both environments substantially reduced mean egg production, but the reduction was greatest under continuous darkness. Under continuous darkness, the distribution of laying times was flattened and shifted toward earlier laying times. Intermittent light of one minute per hour resulted in an almost uniform distribution of laying times throughout the day. In both the continuous darkness and intermittent light environments, selection increased egg production and decreased oviposition interval. However, when compared under a 24-hour-day environment, the lines selected under the continuous darkness and intermittent light environments did not perform as well as a line selected in the 24-hour-day environment. Based on present evidence, there seems to be no benefit to selection under continuous darkness or under intermittent light of one minute per hour.

The effects of both ahemeral and continuous light on ovulation have been studied quite extensively in chickens. Also, selection studies have been conducted in both environments although ahemeral lighting cannot properly be deemed to be a single environment.

Short ahemeral days (light-dark cycles) of less than 22.5 hours can result in significantly reduced egg production. The cause can be traced to the fact that the average length of the ovulation interval in most populations is more than 24 hours. Therefore there is an increase in the number of days that hens must skip in egg laying to adjust from time to time to the imposed oviposition interval (i.e. the number of clutches increase and clutch size decreases; see Morris, 1973 and Biellier, 1985 for a detailed discussion). However, selection for egg production under a short ahemeral day should modify this result, because the distribution of ovulation intervals is modified. Such selection under short ahemeral days has been tested by Marks et al. (1968), and Cahaner and Abplanalp (1979) selecting for egg number, and Foster (1981a) selecting for number of ovulations. In all three studies, selection effectively improved the trait selected. In the studies of Cahaner and Abplanalp (1979) and Foster (1981a), the oviposition interval was reduced. Biellier (1985) reported a short-term rate of egg production greater than 100 percent based on a calendar day by a flock of chickens bred for short ahemeral days and tested under a 23-hour light-dark cycle.

Under continuous light, the oviposition pattern of a flock of hens is much more variable than under a 24-hour day (Morris, 1961; Yoo et al., 1986). Yoo et al. (1986) speculated that without the diurnal control, hens will ovulate according to a natural endogenous cycle unrelated to time of day. However, oviposition time still seemed to be influenced by some rhythmic effect (Morris, 1961; Yoo et al., 1986). Selection effectively reduced the oviposition interval under continuous light (Yoo et al., 1984, 1986). When populations with reduced oviposition intervals were tested under 24-hour days, the number of birds with 24-hour intervals was increased compared to populations not selected for a shorter average interval (Yoo et al., 1986). This would tend to support the hypothesis of an underlying genetic distribution whose expression was limited by the 24-hour-day environment.

Both short ahemeral days and continuous light are promising environments under which to select hens for improved egg production. For both of these environments, many questions remain with regard to techniques and physiological effects, and the question as to which is the best alternative is not fully settled. Chickens selected for a short cycle under continuous or ahemeral light would have to retain this trait in commercial strain crosses to be of practical use in the large areas of the world where chickens are reared and housed in open buildings.

MATHEMATICAL MODELS OF EGG PRODUCTION

Mathematical models of the egg production curve are of potential use to managers and scientists. They can be used to forecast income and flock performance, to evaluate theoretical expectations, or to predict whole-record performance from part-record egg production. The prediction of whole-record egg production requires that the model will reliably fit data from groups of hens (flocks, strains, sire and dam families) and also from individual birds.

Several types of models have been fitted to group or flock data: a compartmental model developed by McMillan (Gavora et al., 1971), a gamma-type function developed by Wood (Timmermans, 1973), an algebraic function consisting of a growth curve and a linear degradation component (Adams and Bell, 1980), a 'logistic' model combining elements of the Adams-Bell and compartmental models (Cason and Britton, 1988), an extended compartmental model using a logistic estimate of the AFE distribution (Yang et al., 1989), and linear regression (Gavora et al., 1982). For egg production to 50-52 weeks of production (or approximately 70-72 weeks of age; whole-record), the compartmental model, the extended compartmental model, the Wood model, the Adams-Bell model, and the logistic model all provide adequate fits to data (Adams and Bell, 1980; Gavora et al., 1971, 1982; McMillan et al., 1986; Cason and Britton, 1988; Yang et al., 1989), as does linear regression fitting only postpeak egg production (McMillan et al., 1989b).

All models do not fit all data equally well. Fitting the compartmental model, the Wood model, and postpeak linear regression based on several part-record lengths, McMillan et al. (1986) found all models predicted whole-record egg production of two selected strains better than that of an unselected control strain, and the rank of the two best predictive models changed with strain. For purposes of forecasting flock income or performance, all models (compartmental, extended compartmental, Wood, Adams-Bell, and logistic models, and postpeak linear regression) should be adequate. The choice of model may depend on other factors, such as simplicity of calculation and the type of data upon which the model must be based. Where the data cannot be synchronized for sexual maturity, the Adams-Bell, logistic, and extended compartmental models are preferable because they fit the sigmoid rise to peak production resulting from differences in AFE (Cason and Britton, 1988); however, when records are synchronized for AFE, the compartmental model may be more desirable. For theoretical purposes, a form of the compartmental model may be preferable due to the biological meaning of its parameters. One model may not necessarily be best in all circumstances or with all data. The properties of the models and the data should be examined, and the appropriate model chosen.

In theoretical and breeding applications, mathematical models of egg production could be used to improve the accuracy of prediction of whole-record egg production for selection purposes or to explore breeding strategies, such as the optimum length of the part-record used for selection. For these purposes, it would be preferable to have a model that could reliably predict egg production of small groups, such as dam or sire families, or best of all, a model that could be used for individual hens. Gavora et al. (1982) fitted the compartmental model, the Wood model, and linear regression on individual and group egg production of survivors synchronized for sexual maturity. The compartmental model provided the best fit for both individual and group egg production, and although the coefficient of determination (R^2) was higher for group than individual data (.97 vs. .71), the fits for individuals were good enough to be potentially useful. More work needs to be done on mathematical models of egg production. In the future, the primary trait of selection for hens may be whole-record egg production estimated from part-record information using a mathematical model (McMillan et al., 1989b).

HERITABILITIES AND GENETIC RELATIONSHIPS

In summarizing the heritabilities and genetic correlations from the literature, each estimate was treated as a datum for the purpose of calculating the mean and variation of these estimates. The precision of each estimate was not considered although some estimates were not very precise. Thus, the standard errors of the heritabilities and genetic correlations reflect variation among the estimates rather than the precision of estimation. This procedure has more validity when the number of estimates of a trait is large.

For the most part, hen-day rate of egg production from the first egg was used as the rate trait. However, some of the literature is deficient in reporting precise trait definitions, and some estimates from housing have been included. For production periods that start after all hens are sexually mature, there is no difference between the two definitions.

Finally, the part-production periods as listed can only be regarded as approximate due to the great variability of record lengths and definitions in the literature. For example, if egg production was recorded from housing to 40 weeks or from housing to 38 weeks, it was included with the category listed under housing to about 39 weeks of age. Similarly, if egg production was reported from 41-53 weeks or from 39-56 weeks, it was summarized with the class of approximately 40-55 weeks of age. Egg production periods too deviant from the ranges listed were not included in these summaries. The period codes and length of the egg records are summarized below:

Period	Code[1]	Approximate record length
Early part record	1	Housing to about 40 weeks of age
Middle part record	2	About 40-55 weeks of age
Final part record	3	About 56-72 weeks of age
Long part record	4	Housing to about 55 weeks of age
Whole record	5	Housing to about 72 weeks of age

[1] The period code is used with each egg production trait, such as hen-housed egg production (HHP), survivor egg production (SEP), hen-day rate of egg production from age at first egg (HDR), to indicate the way egg production was measured and the period over which it was measured.

Heritabilities Of Measures Of Egg Production

Heritabilities based on sire (h^2_S) and sire-plus-dam (h^2_{S+D}) variance components, and parent-offspring regression (h^2_{bop}), and realized heritabilities summarized from the literature are presented in Tables 29.1 to 29.3. There is surprisingly good agreement among the estimates (h^2_S, h^2_{S+D}, h^2_{bop} and realized), and where they differ substantially, it usually results from one or two estimates of the less frequent type that deviate from the norm or

Table 29.1. Heritabilities and standard errors of estimates for hen-housed egg production: from housing to about 39 weeks (HHP1); from housing to about 55 weeks (HHP4); and from housing to about 71 weeks (HHP5); from the literature[1].

		HHP1							
Breed[2]		n[3]	h^2_S	n	h^2_{S+D}	n	h^2_{bop}	n	Realized
White Leghorn	Unsel	25	.29 ± .02	9	.26 ± .03	1	.10		
White Leghorn	Sel	61	.20 ± .01	22	.23 ± .02	4	.27 ± .02	8	.17 ± .04
Other		21	.21 ± .03	9	.26 ± .12	1	.20	8	.13 ± .07

		HHP4				
Breed		n	h^2_S	h^2_{S+D}	h^2_{bop}	Realized
White Leghorn	Unsel	4	.22 ± .00			
White Leghorn	Sel	12	.11 ± .01	.19		
Other		12	.22 ± .04	.44 ± .07		.49 ± .15

		HHP5				
Breed		n	h^2_S	h^2_{S+D}	h^2_{bop}	Realized
White Leghorn	Unsel	12	.18 ± .03	.28 ± .03	.28 ± .01	
White Leghorn	Sel	26	.13 ± .02	.17 ± .04	.25 ± .01	
Other		9	.15 ± .04	.21 ± .08	.44	.28 ± .03

[1] Abd-el-Gawad (1975), Abd-el-Gawad and El-Ibiary (1971), Atalla et al. (1983), Ayyagari et al. (1980, 1983, 1985), Calhoon and Bohren (1974), Chaudhary et al. (1975, 1986), Craig et al. (1969), Dey et al. (1986), Dhaliwal and Acharya (1972), Dickerson (1964), Dixit et al. (1986), El-Hossari (1970a, 1970b, 1971, 1974), Emsley et al. (1977), Engstrom et al. (1986), Flock (1977), Foster (1981a), Goher et al. (1978a, 1978b), Gowe (1977), Gowe and Fairfull (1984, 1985, 1986, unpublished), Hanafi and El-Labban (1984), Kalita and Das (1986), Kinney (1969), Lowe and Garwood (1980), Marks et al. (1968), McClung et al. (1976), Morris (1963), Natarajan and Rathnasabapathy (1978), Nayak and Mishra (1985), Nordskog et al. (1974), Pirchner and von Krosigk (1973), Poggenpoel and Erasmus (1978), Quadeer et al. (1977a, 1977b), Sharma et al. (1984), Shrivastava et al. (1986), Silva et al. (1976), Singh and Mohanty (1985), Singh et al. (1986), Venkatramaiah et al. (1986), von Krosigk (1975), Weyde and Liljedahl (1984), Yeo and Ohh (1982), Yoo et al. (1983).

[2] Unsel = unselected (control) strains, Sel = selected strains.

[3] Number of observations.

Table 29.2. Heritabilities and standard errors of estimates for survivor egg production: from housing to about 39 weeks (SEP1), from about 39 to 55 weeks (SEP2), from about 56 to 71 weeks (SEP3), from housing to about 55 weeks (SEP4), and from housing to about 71 weeks (SEP5); from the literature.

		SEP1				SEP2			
Breed[1]		n[2]	h^2_S	h^2_{S+D}	h^2_{bop}	n	h^2_S	h^2_{S+D}	h^2_{bop}
White Leghorn	Unsel	9	.32±.06	.28±.05		4	.15±.02	.20±.02	
White Leghorn	Sel	7	.21±.03	.23		60	.22±.02		
Other		7	.27±.04	.21±.03	.26±.16	2	.16±.00		

		SEP3				SEP4			
Breed		n	h^2_S	h^2_{S+D}	h^2_{bop}	n	h^2_S	h^2_{S+D}	h^2_{bop}
White Leghorn	Unsel	28	.19±.04			8	.18±.02	.29±.01	
White Leghorn	Sel	60	.20±.01			60	.26±.02		
Other		2	.16±.01			12	.22±.02	.21±.02	.26

		SEP5			
Breed		n	h^2_S	h^2_{S+D}	h^2_{bop}
White Leghorn	Unsel	13	.13±.02	.28±.03	.22
White Leghorn	Sel	8	.28±.03	.17±.02	.57
Other		16	.26±.04	.28±.04	.23±.04

[1] Unsel = unselected (control) strains, Sel = selected strains.
[2] Number of observations.

originate from a different base stock. Usually h^2_{S+D} estimates are larger than h^2_S, but there are frequent exceptions.

The references from which the estimates in Tables 29.1 to 29.12 were obtained are listed under Table 29.1. If an author had estimates of more than one selected or unselected strain, they were each included separately. Estimates from the references cited by Kinney (1969) were also included individually, but the references are not listed here to save space. The numbers of estimates included in each mean varied. There was a great disproportion between breeds in the number of parameter estimates available. It is unfortunate that more research on the genetics of egg production for the breeds and strains that produce brown eggs

Table 29.3. Heritabilities and standard errors of estimates for hen-day rate of egg production: from housing to about 39 weeks (HDR1), from about 39 to 55 weeks (HDR2), from about 56 to 71 weeks (HDR3), from housing to about 55 weeks (HDR4), and from housing to about 71 weeks (HDR5); from the literature.

	HDR1					HDR2			
Breed[1]	n[2]	h^2_S	h^2_{S+D}	h^2_{bop}	Realized	n	h^2_S	h^2_{S+D}	h^2_{bop}
White Unsel Leghorn	13	.21±.03	.06±.11			7	.24±.04		
White Sel Leghorn	45	.12±.01	.17±.02	.10±.03		16	.22±.02		
Other	12	.13±.04	.19±.04	.08±.04		6	.47±.16	.47±.09	.25±.05

	HDR3					HDR4			
Breed	n	h^2_S	h^2_{S+D}	h^2_{bop}	Realized	n	h^2_S	h^2_{S+D}	h^2_{bop}
White Unsel Leghorn	7	.17±.03				4	.37±.05		
White Sel Leghorn	14	.20±.02				11	.20±.02		
Other			.39±.01	.14±.07		2	.17±.05	.20	

	HDR5				
Breed	n	h^2_S	h^2_{S+D}	h^2_{bop}	Realized
White Unsel Leghorn	8	.27±.04			
White Sel Leghorn	27	.21±.01	.16±.01	.12	.04
Other	5	.19±.03	.35±.07	.20±.14	

[1] Unsel = unselected (control) strains, Sel = selected strains.
[2] Number of observations.

has not been published. These birds make up a substantial proportion of the egg production birds of the world and in some countries they are the only laying hens of consequence.

The number of observations included in each of the parameter means displayed is shown for HHP1 in Table 29.1. Heritability estimates for subsequent traits show only the numbers for h^2_S estimates.

Although there were too few observations to justify presenting estimates for brown egg breeds separately, there was no reason to suggest that the heritabilities would be greatly different from the extensive Leghorn data. For example, the two observations found for

Table 29.4. Genetic correlations and standard errors of estimates among hen-housed egg productions and for hen-housed egg production (HHP) with hen-day rate of egg production (HDR) traits[1], from the literature.

Stock[2]	HHP2	HHP3	HHP4	HHP5	HDR1	HDR2	HDR3	HDR4	HDR5
HHP1 WL Unsel	.62±.08	.84±.16	.93±.04	.87±.06	.55±.33	.39±.15	-.21±.61	.59±.12	.49±.15
HHP1 WL Sel	.46±.06	.28±.08	.81±.02	.66±.04	.59±.07	.25±.12	.11±.13	.47±.07	.34±.08
HHP1 Other	.27±.15	.52	.88±.08	.96±.10	.74				.78±.04
HHP2 WL Unsel		1.20±.13	.89±.03	.97±.01	.10±.51	1.01±.01	.94±.13	.93±.10	1.01±.13
HHP2 WL Sel		.90±.04	.91±.09	.97±.02	.74±.07	1.00±.00	.85±.04	1.00±.04	.99±.03
HHP2 Other		1.09±.39	.90						1.11±.19
HHP3 WL Unsel			1.13±.15	1.10±.10	-.09±.74	1.08±.08	1.10±.08	.82±.12	1.03±.10
HHP3 WL Sel			.73±.11	.88±.04	.45±.06	.85±.03	1.02±.02	.80±.06	1.00±.08
HHP3 Other									.81
HHP4 WL Unsel				1.00±.02	.29±.53	.88±.06	.41±.44	.97±.12	.93±.16
HHP4 WL Sel				.94±.04	.57±.06	.79±.04	.65±.04	.48±.07	.82±.03
HHP4 Other				.95±.02	.52			.82	.91±.01
HHP5 WL Unsel					-.41±1.03	1.08±.05	.72±.22	1.01±.13	1.09±.16
HHP5 WL Sel					.67±.05	.93±.02	.90±.01	.92±.03	.95±.02
HHP5 Other									.94±.01

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[2] WL = White Leghorn, Unsel = unselected (control) strains, Sel = selected strains.

Table 29.5. Genetic correlations and standard errors of estimates among survivor egg productions and for survivor egg production (SEP) with hen-day rate of egg production (HDR) traits[1], from the literature.

Stock[2]	SEP2	SEP3	SEP4	SEP5	HDR1	HDR2	HDR3	HDR4	HDR5
SEP1 WL Unsel	.20±.24	-.14±.22	.85±.05	.63±.10	.69±.08	.21±.26	-.15±.23	.53±.15	.09±.30
SEP1 WL Sel	.20±.06	.07±.05	.79±.04	.53±.04	.54±.04	.19±.06	.07±.05	.38±.05	.26±.05
SEP1 Other	.77	.48±5		.58±.07	.30	.20		.70	
SEP2 WL Unsel		1.35±.18	.79±.06	1.00±.06	.69±.18	1.00±.00	1.37±.19	.99±.02	1.13±.05
SEP2 WL Sel		.81±.05	.72±.03	.86±.02	.69±.05	1.00±.00	.81±.02	.95±.01	.95±.01
SEP2 Other		.66±.06	.83±.02	.77					.85
SEP3 WL Unsel			.80±.10	.93±.05	.40±.09	1.37±.20	1.00±.00	.86±.29	1.11±.08
SEP3 WL Sel			.52±.04	.83±.02	.39±.06	.81±.02	1.00±.00	.69±.03	.92±.01
SEP4 WL Unsel				.93±.02	.72±.18	.80±.07	.81±.10	.87±.08	.87±.06
SEP4 WL Sel				.89±.01	.77±.03	.72±.03	.52±.04	.81±.02	.73±.03
SEP4 Other				.88					
SEP5 WL Unsel					.75±.14	1.02±.07	.94±.05	1.05±.05	.94±.05
SEP5 WL Sel					.68±.04	.86±.02	.83±.02	.87±.01	.93±.01
SEP5 Other								.83	

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[2] WL = White Leghorn, Unsel = unselected (control) strains, Sel = selected strains.

survivor egg production of unselected New Hampshire stock had a mean h^2_S of .34 for SEP1, while that of nine equivalent White Leghorn populations was .32. There were three observations for SEP4 of unselected Light Sussex with a mean h^2_S of .17, compared to .18 for eight unselected White Leghorn populations.

Heritability estimates based upon small or poorly structured populations are very variable, and each individual estimate is of little value. More estimates are available based on sire variance components (h^2_S) than on any other basis. The following discussion of the heritabilities will focus on h^2_S estimates because they are the most numerous and have more estimates based on common stocks than other h^2 estimates. However, the reader should be aware that some of the differences in Tables 29.1 to 29.12 result from disproportionate representation of genotypes.

Hen-housed egg production has lower h^2 than survivor egg production or hen-day rate of egg production. This is to be expected due to the influence of viability on hen-housed egg production. The h^2_S of HHP (Table 29.1), SEP (Table 29.2) and HDR (Table 29.3) have averages as follows:

	Housing to 39 wks (period 1)	Housing to 55 wks (period 4)	Housing to 71 wks (period 5)
HHP	.22	.17	.15
SEP	.27	.24	.22
HDR	.14	.24	.22

Heritabilities differ due to selection history. Estimates from selected strains are generally lower than those from unselected strains (see Table 29.1 to 29.3). Although there may be some differences in h^2 among breeds, the limited number of observations for breeds other than Leghorns make such comparisons difficult. Strain differences in h^2 due to differences in genetic bases within breed are common (see for example Gowe and Fairfull, 1984, 1985). More subtle strain differences exist due to disparities in selection history. In two strains selected from a common genetic base, the strain selected for hen-housed egg production had a higher heritability of hen-day rate of egg production in the part record of selection than the strain selected for rate of lay from AFE (Gowe and Fairfull, 1984). Similar changes were noted in other Leghorn strains derived from different genetic backgrounds (Gowe and Fairfull, unpublished). In all cases, the reciprocal relationship with regard to hen-housed egg production did not hold.

The range of published heritability values is wide: h^2_S for hen-housed egg production varies from .03 to .61 for HHP1, 0 to .45 for HHP4, and -.07 to .42 for HHP5. Similar ranges are found for survivor egg production and for hen-day rate of egg production. Nevertheless, most estimates derived from large populations structured to obtain good estimates were clustered fairly closely to the means presented in the tables. The values presented in Tables 29.1 to 29.3 should be used with caution, particularly where there are a limited number of observations. Also, the selected strains would include some selected for a long period and many others selected for only a few generations. Gowe and Fairfull (1985) reported on four Leghorn strains that had been under long-term selection for part-record egg production. When genetic parameters were calculated over the last ten of 29 or 30 generations, the mean h^2_S for the 40 estimates was .12 for HHP1, .09 for HHP4, and .10 for HHP5. These values are about half the magnitude of the corresponding values shown in

Table 29.1. Gowe and Fairfull (1985) also reported genetic parameters of their control strain. The $h^2{}_S$ for HHP1, HHP4, and HHP5 were .28, .22, and .18, respectively. These values are close to the literature means summarized in Table 29.1.

Genetic Correlations

Genetic correlations (r_g) based on sire components of variance and covariance summarized from the literature are presented in Tables 29.4 to 29.12. The r_g values are much more variable than heritabilities. Part of this is due to the fact that genetic correlations are generally estimated with less precision than heritabilities. Correlations with very lowly heritable traits, especially, result in large standard errors of r_g values and the likelihood of results near 1.0 or -1.0. Also, different stocks have unique genetic relationships among traits and these differences can be large. Two strains may have strong relationships between a pair of traits that are in opposite directions, suggesting linkage. Nevertheless, there are general relationships that hold for most populations; for example, egg weight is negatively correlated with egg production in most populations.

Frequently, r_g values of selected strains are not the same as those of the control strains from which they were derived (see Tables 29.4 to 29.12).

Genetic relationships among measures of egg production. Genetic correlations among different measures of egg production are presented in Tables 29.4 to 29.6. Some of these genetic correlations are part-whole correlations, whereby the data of the 'part' are an integral piece of the 'whole', resulting in a substantial built-in correlation even if the distinct parts are not related. For example, a correlation between HHP1 and HHP5 (or HHP4) is a part-whole correlation because the data of the part record make up a substantial portion of egg production of the whole record. Correlations between different egg production traits for which the underlying data spring from a similar root might be viewed as a similar case.

Part-part correlations. The part-part correlations were variable (Tables 29.4 to 29.6). The r_g of period 1 with period 2 for all observations (data for selected, unselected, and others pooled) was highest for HDR and lowest for SEP, while the r_g of HHP1 with HHP2 was intermediate:

	HHP	SEP	HDR
Period 1 with 2	.68	.20	.49
Period 1 with 3	.42	.04	.43
Period 2 with 3	.98	.93	.95
Period 3 with 4	.83	.59	.73

The r_gs of early part record (period 1) with the last third of the record (period 3) were generally the lowest, and the r_gs of period 2 with period 3 part records were the highest part-part correlations. SEP had generally lower r_gs among part periods than HHP or HDR.

It is of considerable interest to note that r_gs of period 1 (early) with period 2 (middle) and period 3 (final) part records calculated as HHP or HDR are lower in selected than unselected lines, and this tendency is emphasized with regard to early and late production (Tables 29.4 and 29.6). Almost without exception, selection for an early part-record egg

Table 29.6. Genetic correlations and standard errors of estimates among hen-day rate of egg production (HDR) traits[1], from the literature.

Stock[2]	HDR2	HDR3	HDR4	HDR5
HDR1 WL Unsel	.70±.18	.51±.13	.84±.22	.82±.15
HDR1 WL Sel	.67±.03	.40±.04	.88±.10	.71±.03
HDR1 Other	.71±.03	.45	.50±.22	.90
HDR2 WL Unsel		1.37±.24	.97±.02	1.13±.06
HDR2 WL Sel		.82±.01	.95±.01	.96±.00
HDR2 Other		.88	.58	.87
HDR3 WL Unsel			.85±.30	1.12±.08
HDR3 WL Sel			.69±.03	.91±.02
HDR3 Other				.65
HDR4 WL Unsel				.98±.01
HDR4 WL Sel				.94±.01
HDR4 Other				.91

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).
[2] WL = White Leghorn, Unsel = unselected (control) strains, Sel = selected strains.

production trait was included in the criteria of the selected lines. Thus, it appears that selection for early part-record egg production may lower its genetic relationships with the residual parts, especially the r_g of early with late part record production (see also the section on *Changes in Genetic Parameters*).

Part-whole correlations. As a group the part-whole correlations were high (Tables 29.4 to 29.6). When all observations were averaged, the lowest r_gs were those of period 1 with period 5. The remaining r_gs were higher:

	HHP	HDR	SEP
Period 1 with 4	.84	.81	.85
Period 1 with 5	.73	.74	.56
Period 2 with 4	.91	.74	.95
Period 2 with 5	.97	.90	1.00
Period 3 with 5	.93	.86	.96
Period 4 with 5	.95	.92	.95

As would be expected from the trend in part-part r_g, part-whole r_gs for unselected lines were higher than those of selected lines.

Correlations among different measures of egg production. Genetic correlations of HDR with HHP and SEP are presented in Tables 29.4 and 29.5. These r_g values are normally high (.7 or higher). In general, the highest correlations are those of corresponding periods, i.e. HHP2 with HDR2, SEP3 with HDR3, etc. The lowest r_gs involve period 1 HHP, SEP, and HDR with period 2 or period 3 records, or period 1 with period 4 or 5 records. Some of these r_gs are very low or negative, e.g. SEP1 with HDR3, SEP1 with HDR5, HHP1 with HDR3, HDR1 with HHP3, or HDR1 with HHP5.

Relationships of egg production measures with other production traits. Genetic correlations of different measures of egg production with other production traits (hatchability, viability, body weight, age at first egg, egg mass, feed conversion, and residual feed consumption) are presented in Tables 29.7 to 29.9.

All valid egg production records (EP) and HDR traits appear to be negatively correlated with viability traits (Tables 29.7 and 29.9), although a few estimates in unselected lines were positive. However, the standard errors of these r_g estimates were very large, indicating that the estimates were extremely variable, possibly due to the low heritability of viability. These correlations are of little value and suggest that selection programs should be carried out under environments with very low levels of mortality.

For rearing body weights (8-12 weeks of age), the few genetic correlations with egg production traits seem to be generally positive, although the r_gs of egg production with mature body weights were mostly negative (Tables 29.7 to 29.9). The r_gs of body weights at 18-22 weeks of age with egg production traits were positive for early part-record egg production and lower or increasingly negative with later parts:

	Period 1	Period 2	Period 3	Period 4	Period 5
EP	.29	-.02	-.16	.04	-.07
SEP	.24	-.04	-.02	.13	.07
HDR	.13	-.05	-.02	.04	-.11

The r_gs of mature body weights (40-52 weeks of age) with SEP traits were low and negative, while mature body weights and HDR traits had high negative r_g values for the early part-record and low negative r_g values for the later periods:

	Period 1	Period 2	Period 3	Period 4	Period 5
SEP	-.08	-.01	-.05	-.08	-.06
HDR	-.43	-.03	-.05	.05	-.06

The r_gs of mature body weights with EP traits followed two distinct trends in Leghorn stocks (Table 29.7). In unselected strains, the r_gs were positive, ranging from .38 to .45. In selected strains, the r_gs of mature body weights were negative and low with the early part-record production (EP1), and large and negative with the late part record (EP3).

The r_gs of age at first egg with EP and SEP were large and negative for early part-record production, but low and positive with later part records (see below). The r_gs of age at first egg with HDR were lower, but followed the same general trend:

	Period 1	Period 2	Period 3	Period 4	Period 5
EP	-.71	.06	.06	-.46	-.26
SEP	-.85	.05	.13	-.54	-.30
HDR	-.17	.09	.08	-.03	.10

Table 29.7. Genetic correlations and standard errors of estimates of egg production[1] (EP) traits[2] with hatchability, viability, body weight, age at first egg (AFE), egg mass, feed conversion (FConv)[3], and residual feed consumption (RFC)[3], from the literature.

	Hatchability	Viability		Body weight				Egg mass to			
Stock[4]		to 273d	to 497d	8-12wk	18-22wk	40-52wk	AFE	273d	497d	FConv[5]	RFC[5]
EP1 WL Un		-.43±.23	.58±1.42		.23±.14	.45±.19	-.85±.20	.92±.01		-1.21	-.04±.32
EP1 WL Sel		-.22±.18	-.55±.13		.43±.10	-.08±.07	-.68±.12	.93±.01		.10±.15	.05±.11
EP1 Other					-1.04±.72	-.31±.89	-.62±.11				
EP2 WL Un		-.45±.50	-1.90±.83		.11±.12	.38±.28	-.10±.19			-.90	.10±.14
EP2 WL Sel		-1.11±1.00	-.27±.21		-.05±.06	-.01±.10	.11±.07			-.27±.24	.12±.24
EP3 WL Un		-.86±.23	-1.22±.48		-.10±.26	.64±.26	.02±.24			-.96	-.13±.07
EP3 WL Sel		-.12±.21	-.15±.18		-.17±.07	-.42±.29	.07±.07			-.77±.19	-.36±.16
EP4 WL Un		-.33±.56	.54±1.55		.21±.08	.43±.21	-.55±.25			-1.08	.13±.21
EP4 WL Sel		-.65±.38	-.50±.18		.09±.06	-.12±.16	-.43±.08			-.06±.25	.10±.21
EP4 Other	.23		-.56	-.17	-1.05±.20	-.14±.07	-.57±.08				
EP5 WL Un		-1.43±.95	-1.01±.22		.14±.08	.44±.16	-.35±.24			-1.04	-.06±.18
EP5 WL Sel		-.75±.46	-.60±.12		-.03±.06	-.54±.56	-.20±.09			-.53±.15	-.18±.14
EP5 Other					-1.10±.13	-.47±.06	-.50±.13		.76		

[1] All hens with a valid observation for egg production and the other trait in the correlation.

[2] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[3] Measured postpeak for a four-week period.

[4] WL = White Leghorn, Un = unselected (control) strains, Sel = selected strains.

[5] From Gowe and Fairfull (unpublished).

Table 29.8. Genetic correlations and standard errors of estimates of survivor egg production (SEP) traits[1] with hatchability, viability, body weight, age at first egg (AFE), and egg mass, from the literature.

Stock[2]	Hatchability	Viability to 497d	Body weight 8-12wk	Body weight 18-22wk	Body weight 40-52wk	AFE	Egg Mass to 273d
SEP1 WL Un				.24±.09	.03±.10	-.91±.06	
SEP1 WL Sel				.26±.04	-.10±.04	-.86±.02	
SEP1 Other			.12±.04	.07±.01	-.26±.11	-.62±.15	.91
SEP2 WL Un				-.25±.32	-.07±.12	.03±.25	
SEP2 WL Sel				.01±.04	.00±.05	.10±.05	
SEP2 Other			.04±.07	.03±.04	.09	-.41±.22	
SEP3 WL Un				.05±.16	-.03±.15	.14±.24	
SEP3 WL Sel				-.04±.05	-.06±.05	.13±.04	
SEP3 Other						-.15	
SEP4 WL Un				-.03±.28	-.10±.13	-.59±.10	
SEP4 WL Sel				.17±.05	-.08±.04	-.52±.04	
SEP4 Other	.30		-.02±.12		.03±.03	-.58	
SEP5 WL Un				.09±.11	.00±.10	-.46±.10	
SEP5 WL Sel				.08±.05	-.08±.05	-.26±.04	
SEP5 Other		.20±.33	.10±.01	-.05±.08	.03	-.36	

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).
[2] WL = White Leghorn, Un = unselected (control) strains, Sel = selected strains.

Table 29.9. Genetic correlations and standard errors of estimates of hen-day rate of egg production (HDR) traits[1], with hatchability, viability, body weight, age at first egg (AFE), egg mass, feed conversion (FConv)[2], and residual feed consumption (RFC)[2], from the literature.

	Hatchability	Viability		Body weight				Egg mass to			
Stock[3]		to 273d	to 497d	8-12wk	18-22wk	40-52wk	AFE	273d	497d	FConv[4]	RFC[4]
HDR1 WL Un		.04±.63	.83±2.32		.22±.11	-1.46±1.28	-.09±.35			-1.34	.55±.69
HDR1 WL Sel		-.47±.22	-.49±.19	.16±.23	.11±.05	-.13±.09	-.23±.07	.78±.05		.16±.26	-.09±.19
HDR1 Other				-.12	-.15	-.60±.27	.21±.19				
HDR2 WL Un		-1.32±.73	-2.24±.83		-.24±.33	-.06±.12	.00±.28			-.88	.01±.04
HDR2 WL Sel		-.83±.75	-.18±.20		.01±.04	.00±.05	.12±.07			-.28±.22	.07±.22
HDR2 Other				.40±.18	-.22±.26	-.24±.14	.02				
HDR3 WL Un		-.68±.19	-.98±.44		.06±.16	-.02±.15	-.05±.24			-.93	-.07±.09
HDR3 WL Sel		-.12±.23	-.08±.18		-.04±.05	-.06±.05	.12±.04			-.75±.17	-.38±.16
HDR3 Other				.01	-.09	.00					
HDR4 WL Un		-.33±.88	-1.23±.92		.19±.26	.30±.25	-.05±.26			-1.06	-.09±.08
HDR4 WL Sel		-.78±.52	-.26±.20		-.01±.04	-.02±.05	-.02±1.59			-.14±.25	.10±.24
HDR4 Other	.31		-.27	-.47±.21	.56	.05±.08	-.19				
HDR5 WL Un		-1.09±.68	-1.37±.52		-.41±.38	-.10±.12	.45±.79			-.92	-.05±.06
HDR5 WL Sel		-.86±.62	-.22±.23		-.02±.04	-.06±.08	.01±.03			-.55±.14	-.22±.17
HDR5 Other			.42	.11	-.13±.02	.03±.10	-.02		.70		

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[2] Measured postpeak for a four-week period.

[3] WL = White Leghorn, Un = unselected (control) strains, Sel = selected strains.

[4] From Gowe and Fairfull (unpublished).

It is obvious that no matter how early part-record egg production is measured, genetically the hens that mature early produce the most eggs in that early period.

The few r_g estimates of egg mass with egg production traits were high and positive (Table 29.7 to 29.9). The r_gs of residual feed consumption (feed consumed corrected for production of egg mass, body weight, and change in body weight) with EP and HDR are shown in Tables 29.7 and 29.9.

Relationships of egg production measures with egg quality traits. Genetic correlations of different measures of egg production with egg quality traits (egg weight, egg specific gravity, Haugh units, egg shell shape, blood spot incidence, egg shell texture, egg shell color, and incidence of intact eggs) are presented in Tables 29.10 to 29.12.

Egg weight (or egg size) is one of the most important quality traits and one of the most uniform with regard to relationships with egg production measures. EP, SEP, and HDR were negatively correlated with egg weight (periods 1, 2 and 3) with r_gs mostly in the range of -.2 to -.6 (Tables 29.10 to 29.12). There was no apparent trend with regard to age nor did there appear to be a systematic difference between unselected and selected stocks.

Egg specific gravity (SG, an indirect measure of egg shell strength) had moderate to low genetic correlations with SEP and HDR that were mostly negative (Tables 29.11 and 29.12). There was possibly a trend for these r_gs to become lower and turn positive for SEP3 and HDR3. Also, there were some apparent differences between estimates in unselected and selected lines. The r_gs of Haugh units (albumen height corrected for egg size, a measure of internal egg quality) with SEP and HDR were similar in size and trend to those noted above for SG; however, the correlations with longer-record productions (periods 4 and 5) tended to be positive and very low.

Egg shell shape had mostly negative r_gs with SEP and HDR, normally in the range of -.1 to -.3 (Tables 29.11 and 29.12). However, early SEP seemed to be positively correlated with early and late shell shape. With the exception of the early egg production record, these r_gs of selected strains were apparently lower than those of unselected strains and were close to zero.

The r_gs of blood spot incidence (a measure of internal egg quality) with SEP and HDR were generally negative or low (Tables 29.11 and 29.12). Some of these r_gs were very high, but the higher r_gs had large standard errors indicating a great deal of variation among estimates. The r_gs of egg shell texture with SEP and HDR ranged from -.17 to .25 with slightly more of the estimates being positive.

Summary

The h^2s of HHP, SEP and HDR were generally comparable, except that HDR had generally lower values than HHP and SEP for the early part record (period 1), and HHP had lower values than SEP and HDR in periods 4 and 5. Also, selected strains had lower h^2s estimates than those of unselected control strains.

The r_gs among records for each egg production were generally very high for part-whole correlations, but lower for part-part correlations, especially the r_gs of period 1 with 3. In fact for some selected strains, the r_gs of periods 1 and 3 were negative. Also, the r_gs among part records for SEP were very low, much lower than comparable values for HHP and HDR, indicating that SEP may be the least desirable of the three traits with which to

Table 29.10. Genetic correlations and standard errors of estimates of egg production[1] (EP) traits[2] with egg weight, egg specific gravity, Haugh units, shell shape, blood spots, shell texture, shell color, and percentage intact egg shells, from the literature.

	Egg weight			Specific gravity		Haugh units		Shell shape		Blood spots	Shell text	Shell color	Percent intact	
Stock[3]	20-39[4]	40-55[4]	56-72[4]	20-39[4]	40-55[4]	20-39[4]	40-55[4]	20-39[4]	40-55[4]	20-39[4]	20-39[4]	20-39[4]	20-39[4]	56-72[4]
EP1 WL Un	-.18±.11													
EP1 WL Sel	-.39±.06	-.32±.06	-.62		.51		-.37				-.19		-.02	.67
EP2 WL Un		-.16												
EP2 WL Sel		-.55±.01												
EP3 WL Sel	-.51		-.59										-.30	-.13
EP4 WL Sel		-.53			.15		-.18		.31					
EP4 Other														
EP5 WL Sel	-.31	-.40		-.26	-.19	-.06	-.02	-.09		-.04	.02	-.04		
EP5 Other	-1.01±.35		-.51±.10											

[1] All hens with a valid observation for egg production and the other trait in the correlation.

[2] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[3] WL = White Leghorn, Un = unselected (control) strains, Sel = selected strains.

[4] Age range in weeks.

Table 29.11. Genetic correlations and standard errors of estimates of survivor egg production (SEP) traits[1] with egg weight, egg specific gravity, Haugh units, shell shape, blood spots, and shell texture, from the literature[2].

	Egg weight			Specific gravity		Haugh units		Shell shape		Blood spots		Shell texture
Stock[3]	20-39[4]	40-55[4]	56-72[4]	20-39[4]	56-72[4]	20-39[4]	56-72[4]	20-39[4]	56-72[4]	20-39[4]	56-72[4]	56-72[4]
SEP1 WL Un	-.18±.10		-.10±.07	-.07±.10	-.12±.19	-.12±.12	-.16±.10	.09±.13	.05±.11	.03±.27	.10±.23	.18±.14
SEP1 WL Sel	-.31±.04		-.28±.04	-.20±.03	-.21±.05	-.10±.04	-.08±.04	.13±.04	.09±.04	.07±.09	-.03±.08	.16±.05
SEP1 Other	.20±.02	-.12±.03		.09		-.20		-.08±.14				
SEP2 WL Un	-.43±.14		-.39±.17	.00±.10	-.17±.23	-.18±.13	.04±.12	-.30±.13	-.54±.16	-.92±.42	-.58±.42	-.11±.22
SEP2 WL Sel	-.17±.04		-.44±.03	-.11±.04	-.18±.04	.13±.05	.16±.05	-.11±.05	-.05±.05	.04±.10	-.22±.08	.14±.05
SEP2 Other	.20±.05	-.28±.06										
SEP3 WL Un	-.28±.17		-.34±.14	.11±.10	-.22±.30	-.17±.19	.18±.10	-.15±.21	-.37±.12	-.02±.25	.46±.93	-.02±.30
SEP3 WL Sel	-.15±.04		-.35±.03	.09±.04	.10+.05	.10±.05	.16±.05	-.10±.04	-.10±.04	.01±.09	-.22±.07	-.15±.05
SEP4 WL Un	-.38±.12		-.32±.10	-.18±.15	-.15±.19	-.06±.14	-.04±.10	-.23±.18	-.31+.14	-.43±.38	-.07±.17	.09±.20
SEP4 WL Sel	-.32±.04		-.45±.04	-.21±.04	-.24±.04	.00±.04	.04±.04	.02±.05	.00±.04	.06±.08	-.14±.08	.19±.05
SEP5 WL Un	-.38±.12		-.31±.10	-.04±.11	-.31±.20	-.06±.11	.01±.11	-.26±.13	-.35±.10	-.12±.13	-.08±.35	.02±.24
SEP5 WL Sel	-.27±.04		-.47±.03	-.09±.04	-.09±.04	.06±.04	.11±.04	-.04±.05	-.03±.04	.06±.08	-.18±.07	.03±.04
SEP5 Other	-.11±.06	-.04		-.30		-.32±.03		-.18		.08		

[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[2] With the exception of egg weight, most values are from Gowe and Fairfull (unpublished).

[3] WL = White Leghorn, Un = unselected (control) strains, Sel = selected strains.

[4] Age range in weeks.

Table 29.12. Genetic correlations and standard errors of estimates of hen-day rate of egg production (HDR) traits[1] with egg weight, egg specific gravity, Haugh units, shell shape, blood spots, shell texture, and shell color, from the literature[2].

Stock[3]	Egg weight			Specific gravity			Haugh units			Shell shape			Blood spots		Shell texture		Shell color
	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	56 - 72^4	20 - 39^4	56 - 72^4	20 - 39^4
HDR1 WL Un	-.24 ±.11		-.23 ±.12	-.17 ±.22		-.02 ±.35	-.06 ±.16		.14 ±.14	-.22 ±.15		-.22 ±.25	-.09 ±.40	.15 ±.41		-.17 ±.28	
HDR1 WL Sel	-.31 ±.10		-.41 ±.04	-.21 ±.06		-.22 ±.05	.13 ±.06		.10 ±.05	-.06 ±.06		.03 ±.06	.00 ±.13	-.16 ±.09	.06	.21 ±.05	-.14
HDR1 Other	-.31 ±.10	-.68 ±.18		-.41	-.06 ±.12		.08	.03 ±.24		-.10							
HDR2 WL Un	-.44 ±.15		-.39 ±.15	.00 ±.10		-.18 ±.22	-.17 ±.13		.04 ±.12	-.31 ±.14		-.53 ±.16	-.97 ±.44	-.58 ±.42		-.12 ±.22	
HDR2 WL Sel	-.17 ±.04		-.44 ±.03	-.11 ±.04		-.18 ±.04	.13 ±.05		.17 ±.05	-.11 ±.05		-.05 ±.05	.04 ±.10	-.22 ±.08		.14 ±.05	
HDR2 Other	-.11 ±.05	-.07 ±.10		-.44			.00			.37							
HDR3 WL Un	-.29 ±.17		-.36 ±.15	.12 ±.11		-.21 ±.31	-.19 ±.20		.19 ±.10	-.15 ±.22		-.36 ±.12	-.02 ±.25	.46 ±.93		.01 ±.33	
HDR3 WL Sel	-.15 ±.04		-.35 ±.03	.09 ±.04		.10 ±.05	.10 ±.05		.16 ±.05	-.10 ±.04		-.10 ±.04	.01 ±.09	-.22 ±.07		-.15 ±.05	
HDR3 Other	-.09	-.37		-.25			-.48			.25							
HDR4 WL Un	-.34 ±.12		-.32 ±.13	-.25 ±.18		-.11 ±.19	-.46 ±.37		-.06 ±.13	-.29 ±.19		-.33 ±.22	-.66 ±.36	-.64 ±.41		.25 ±.32	

Continued ...

Table 29.12. Genetic correlations and standard errors of estimates of hen-day rate of egg production (HDR) traits[1] with egg weight, egg specific gravity, Haugh units, shell shape, blood spots, shell texture, and shell color, from the literature[2]. (Continued)

Stock[3]	Egg weight			Specific gravity			Haugh units			Shell shape			Blood spots		Shell texture		Shell color
	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	40 - 55^4	56 - 72^4	20 - 39^4	56 - 72^4	20 - 39^4	56 - 72^4	20 - 39^4
HDR4 WL Sel	-.25 ±.04	-.37	-.47 ±.03	-.16 ±.04	.00	-.21 ±.04	.12 ±.05	-.11	.13 ±.04	-.08 ±.05	.12	-.01 ±.05	.04 ±.10	-.24 ±.08	.09	.18 ±.05	-.08
HDR4 Other	-.23	.09											-.13				
HDR5 WL Un	-.53 ±.15		-.30 ±.10	-.08 ±.17		-.28 ±.19	-.09 ±.19		.03 ±.11	-.35 ±.13		-.43 ±.09	-.66 ±.43	-.22 ±.37		.06 ±.26	
HDR5 WL Sel	-.22 ±.06		-.44 ±.04	-.04 ±.05		-.06 ±.04	.12 ±.05		.16 ±.04	-.09 ±.05		-.04 ±.05	.05 ±.12	-.21 ±.07	.02	.01 ±.05	-.06
HDR5 Other	-.50 ±.09	-.18 ±.13	-.51 ±.14	-.23			-.19 ±.13			.17							

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[1] Period: 1 = housing to 39 weeks (approximately), 2 = 40 to 55 weeks (approximately), 3 = 56 to 71 weeks (approximately), 4 = housing to 55 weeks (approximately), 5 = housing to 71 weeks (approximately).

[2] With the exception of egg weight and specific gravity, most values are from Gowe and Fairfull (unpublished).

[3] WL = White Leghorn, Un = unselected (control) strains, Sel = Selected strains.

[4] Age range in weeks.

attempt to improve whole-record egg production, at least initially.

The r_g of early egg production with AFE was generally negative, but the r_g with HDR was much lower than those for HHP and SEP with AFE. Mature body weight was negatively correlated with egg production, with the exception of HHP in unselected strains. Viability and the egg quality traits had generally negative r_gs with egg production. However, the variability among r_g estimates for viability and blood spot incidence was high, indicating that the estimates probably were not very reliable, possibly because of the low heritability of these traits or low incidence.

SELECTION IN LAYER STOCKS

Selection Studies

Egg production alone. In the great majority of single trait selection experiments, positive genetic progress for the trait selected, egg numbers or rate of egg production, was recorded (Table 29.13). However, in a few cases, genetic progress was absent or negative (Saadeh et al., 1968; Calhoon and Bohren, 1974; Nordskog et al., 1974; Ibe et al., 1982).

Because of the limits imposed by their objectives, correlated responses to selection were not reported from all studies, which greatly diminished their value. Reports of many part-record selection studies failed to include changes (or their absence) in whole-record egg production or rate. Even those which included some correlated responses generally did not publish results on all the traits needed for a complete interpretation of unitrait selection.

Selection for part-record rate resulted in increased whole-record rate in Kinney et al. (1970) and in one line of Nordskog et al. (1974), and increased period 4 egg production (housing to about 55 weeks of age) in one of two lines in Goher et al. (1978b), but no change in period 1 egg production (housing to about 40 weeks) or period 4 rate in Goher et al. (1978b). Selection for part-record egg numbers resulted in improved period 4 egg production and rate (Weyde and Liljedahl, 1984), whole-record egg production (Morris, 1963; El-Hossari 1970b; Poggenpoel and Erasmus, 1978), and part-record rate (Ayyagari et al., 1980; Liljedahl and Weyde, 1980). In the long-term studies of Morris (1963) and Erasmus (Poggenpoel and Erasmus, 1978), egg production in the residual record showed no improvement even though Morris (1963) reported positive response in the first few generations.

Selection for part-record egg numbers or rate decreased egg weight in 20 of 31 cases, only four increased (Table 29.14), and in several studies the reductions in egg size were much greater than expected. Part-record selection for egg production caused AFE to decline in 14 of 29 lines with two instances of later AFEs (Table 29.14). Similarly, mature body weight decreased in 15 of 28 selected lines and increased in only two strains (Table 29.14). Housing body weight decreased in five of ten cases (Table 29.14). Part-record selection for egg production improved egg mass in ten of 12 lines, without any reports of significant declines (Table 29.14). Ayyagari et al. (1982) reported improved feed conversion in four lines from the same genetic base selected for part-record egg production. In the four lines of Morris (1963), Poggenpoel and Erasmus (1978), Kolstad (1980) and Sorensen et al. (1980), viability was not significantly reduced .

Egg shell strength as measured by egg specific gravity, or egg shell thickness, declined in three and remained unchanged in six of nine part-record selected lines (Craig et al., 1969;

Table 29.13. Single trait selection for egg production or rate: selection responses per generation, and ratios of observed and expected (O/E) selection differentials and responses, with a brief description of the experiments from which they derive.

	Selection		Generations				Selection responses		Observed/expected selection			
									Differentials		Responses	
Reference	Trait[1]	Method(s)[2]	of selection	Sires	Dams	Progeny[9]	Mean	Range	Mean	Range	Mean	Range
Ayyagari et al. (1980)	EP 280	Comb[3]	4	36.4	165.7	708.6	2.5 eggs	1.1 to 4.7	1.01	.99 to 1.02	.94	.50 to 1.47
Calhoon and Bohren (1974)	HDR 280	Comb[3], RRS	6	10	50	500	.2%	-.6 to .8 WLS[10,11]			.08	-.28 to .36 WLS[11]
				10	50	1000[12]	-.4%	-1.0 to .4 RRS[10,11]			-.31	-.77 to .34 RRS[11]
El-Hossari (1970a)	EP 240	FS	5			800	2.1 eggs					
Garwood and Lowe (1979)	HDR 260	Ind, HS, Comb	11	-[13]	-[13]	-[13]	.7%	.3 to .9	1.00	.99 to 1.00	.47	.14 to .91
Garwood and Lowe (1981)	HDR 280	HS, Comb[4]	1	6	36	180						
Goher et al. (1978a)	HDR 230	Ind	11	5-11	7-27	140-512[14]	.7%	.2 to 1.2[15]			.54	.46 to .61
Kinney et al. (1970)	HDR 260	Ind, FS, HS, Comb	8	10-32	50-300	400-1200	3.8%	2.7 to 5.3	1.01	1.00 to 1.05	.32 .43	.20 to .43[16] .20 to .84[17]
Kolstad (1980)	EP 294	Comb	4	17-21	72-110	397-474	6.6 eggs				.90	
Liljedahl and Weyde (1980)	EP 294	Comb	4	17-18	115-137	418-522	3.6 eggs				.65	
Morris (1963)	EP[5]	Comb[6,7]	11	6-17	30-109	86-475	2.7 eggs					
Nordskog et al. (1974)	HDR 224	Comb[3]	10	16	7-14	622	.2%	-.4 to .8				

Continued ...

Table 29.13. Single trait selection for egg production or rate: selection responses per generation, and ratios of observed and expected (O/E) selection differentials and responses, with a brief description of the experiments from which they derive. (Continued)

Reference	Selection		Generations				Selection responses		Observed/expected selection			
									Differentials		Responses	
	Trait[1]	Method(s)[2]	of selection	Sires	Dams	Progeny[9]	Mean	Range	Mean	Range	Mean	Range
Poggenpoel and Erasmus (1978)	EP 275[8]	Comb[6]	7	18	107	525	3.0 eggs		.98		.69	
Saadeh et al. (1968)	HDR 260	Comb[3], RRS	7	20	100	500-600	.7%	.3 to 1.3 WLS[11]				
				20	100	500-600[12]	.0%	-.2 to .3 RRS[11]				
Singh and Kumar (1983)	EP 280	Comb	2				5.2 eggs				.74	
Sorensen et al. (1980)	EP 294	Comb	3	20	90-100	350-552	2.9 eggs					
Venkatramaiah et al. (1986)	EP 280	Comb[3]	2	25-30	110-163	319-630	2.2 eggs	1.0 to 3.7	.99	.96 to 1.03		

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[1] EP = egg production, HDR = hen-day rate of egg production from AFE, 224 = part record to 224 days of age, 230 = part record to 230 days of age, 260 = part record to 260 days of age, etc. [2] Ind = individual, FS = full-sib family, HS = half-sib family, Comb = combined (Ind, FS, and HS), and RRS = reciprocal recurrent selection. [3] Osborne Index (Osborne, 1957a, 1957b). [4] Combined individual and half-sib family selection with six replicates. [5] Reduced part-record mean winter pause from 1948 to 1954, part-record mean clutch size in 1955, part-record production index from 1956. The part record terminated on May 31 although there were 3-11 hatches. [6] Combined individual and full-sib family selection. [7] An Osborne Index (Osborne, 1957a, 1957b) was used for the last two generations. [8] Selected on whole-record egg production in the nine preceding generations (1953 to 1962). [9] Hens housed (or pullets). [10] WLS = results for within-line selection, RRS = results for reciprocal recurrent selection. [11] Response in pure lines only. [12] Crossbred progeny, pure line progeny not included. [13] See Kinney et al. (1970). [14] From Ibe et al. (1982). [15] Outbred lines only. [16] Selection differentials and responses of Ind method not included. [17] Selection responses of Ind method included.

Table 29.14. Single trait selection for egg production or rate: directional[1] summary of correlated selection responses.

Reference	Egg weight	AFE	Body weight		Egg mass
			Housing	Mature	
Ayyagari et al. (1980, 1982)	4-	4-	4-	4-	4+
Craig et al. (1969)	2=, 3-	2+, 3=		2=, 3-	
El-Hossari (1970a, 1970b)	1+	1=	1=	1=	
Garwood et al. (1980)	1+, 1=	1=, 1-			
Goher et al. (1978a)	2-	2=		2-	
Kinney et al. (1970)	1=, 4-	5=		1=, 4-	
Kolstad (1980)	1-	1-		1=	1+
Liljedahl and Wyde (1980)[2]	1-	1-			1+
Morris (1963)	1-	1-		1-	
Nordskog et al. (1974)	1=, 1-			2=	2=
Poggenpoel and Erasmus (1978)	1-	1-		1=	
Singh and Kumar (1983)	1-	1-	1=	1-	
Sorensen et al. (1980)	1=	1=		1=	
Venkatramaiah et al. (1986)	2+, 1=, 1-	4-	1+, 2=, 1-	2+, 2=	4+
Total	4+, 7=, 20-	2+, 13=, 14-	1+, 4=, 5-	2+, 11=, 15-	2=, 10+

[1] +, increase; =, no change; -, decrease.

[2] Results over four additional generations are in agreement (Weyde and Liljedahl, 1984)

Goher et al., 1978b; Kolstad, 1980; Sorensen et al., 1980). Albumen height (or Haugh units) decreased in three and increased in one of seven selected lines (Craig et al., 1969; Kolstad, 1980; Sorensen et al., 1980). Only Kolstad (1980) and Sorensen et al. (1980) reported on fertility and hatchability which remained unchanged. The lines of Kolstad (1980) and Sorensen et al. (1980) were derived from the same genetic base and were selected using the same procedures. In a line selected for egg production to 275 days and a daughter line (split off after eight generations) selected for rate to 275 days, Frankham and Weiss (1969) found that the relative adult aggressiveness of the selected lines was equal to or less than that of the unselected control from which they were derived. Craig et al. (1975) demonstrated that the part-record production selected line exhibited more social dominance and aggressiveness than the control as juveniles.

There was very good agreement between observed and expected (predicted) selection differentials (Table 29.13). The mean ratio of observed to expected (O/E) selection differentials per generation was normally in the range of .96 to 1.05. This indicates that selection pressure, and how it was applied, was well estimated within generations. Thus, discrepancies in O/E selection responses do not appear to have their roots in the selection differential.

Observed and expected selection response (gain) normally did not agree as well as for selection differentials (Table 29.13). Observed selection responses per generation were normally lower than expected with O/E in the range of -.77 to 1.47. In two studies (Morris, 1963; Poggenpoel and Erasmus, 1978), a large portion of the discrepancy appeared to be due to poor estimates of additive genetic variation or a poor choice of an estimator, but this does not seem to apply in all cases.

Short-term selection responses appear to be more predictable than long-term response.

This, a trivial observation, is to be expected because genetic drift and genotype-environment interaction would reduce genetic variation among selection candidates due to initial selection, and minor deviations in selection intensity would influence long-term response more than short-term. However, response per generation does not completely depend upon duration of selection, for several short-term experiments were less predictable in this regard than longer studies. Also, genetic parameters may change with selection, so that correlated responses may change as a result of selection; several such changes were reported in different populations.

Several single-trait selection studies were designed to compare selection methods. Both Saadeh et al. (1968) and Calhoon and Bohren (1974) compared within-line selection (WLS) and reciprocal recurrent selection (RRS) with the same inconclusive result (see also Chapter 37). WLS improved pure line performance more than RRS. This coincides with reports from industry that those using RRS have added pure line selection to their RRS programs (Arthur, 1986; Flock, 1988).

A number of experiments using part-record selection for rate were carried out comparing selection based on individual and/or family information (see also **Nonadditive inheritance** for relevant comments). Kinney et al. (1970) compared selection based on individual (Mass), full-sib family (FS), half-sib family (HS), and a combination (Index) of individual, FS and HS records (Index had two lines). In terms of efficiency (O/E selection response), Mass selection was best, followed by Index selection (.84 for Mass vs. .43 and .38 for Index), but this was based mainly on the lower expectations for Mass selection. Both the Mass and Index methods had approximately equal population sizes (pullets housed), and one of the two Index lines had a comparable mating structure to the Mass line. The selection responses to both methods were similar with a slight edge to Index selection (5.3 and 4.9 for Index vs. 4.2 for Mass), so that on the basis of equal resources with roughly the same number and proportion selected, Index selection had a slender lead. Garwood and Lowe (1979) found that HS and Index (HS plus individual information) selection produced greater responses than Mass selection, but this result was reversed for standardized responses (Mass had greater responses than HS and Index). When Garwood et al. (1980) compared Mass and HS selection in a single-generation selection experiment, Mass selection was better before and after standardization. Another single-generation selection experiment compared HS and Index (HS plus individual information) selection, but this study was too small to reasonably expect a significant difference and there was none (Garwood and Lowe, 1981).

In summary, single-trait selection for egg production was generally successful. Although selection differentials were well predicted, genetic gains were usually overestimated. In comparisons of selection schemes, the expected superiority of index selection over mass selection, or family selection over mass selection was usually not realized.

Egg production plus egg weight or egg mass. In the few studies reporting selection for egg production and egg size, egg production was significantly improved as was egg size in the majority of them (Table 29.15). Gains in egg mass were also significant for egg production plus egg weight selected lines. Where comparable lines were selected for egg production alone, the gains per generation for egg production were greater (average of 1.8 eggs more) than that of lines selected for both egg production and egg size, and there was no difference in egg mass. Lines selected for egg mass improved for egg mass, and made correlated gains in egg production and egg weight (Table 29.15). Thus, selection for egg production plus egg weight seems roughly equivalent to selection for egg mass, but

Table 29.15. Selection for egg number plus egg weight or egg mass: selection responses per generation, and ratios of observed and expected (O/E) selection differentials and responses, with a brief description of the experiments from which they derive.

Reference	Selection		Generations				Selection responses			Observed/expected selection				
										Differentials for EM		Responses		
	Trait(s)[1]	Method(s)[2]	of selection	Sires	Dams	Progeny[8]	EP	EW	EM	Mean	Range	EP	EW	EM
Kolstad (1980)	EP 294+EW[3]	Comb	4	13-22	72-108	390-481	3.0 eggs	.4g	167g			1.14	1.15	
Liljedahl and Weyde (1980)	EP 294+EW[3]	Comb	4	17-20	75-136	295-543	2.2 eggs	.4g	151g			1.35	.64	1.10
Marks (1981)	EM 154-294[4]	Comb	3	6	24	100		.9g	1.57g/d	1.00	.99 to 1.01			
Natarajan and Rathnasabapathy (1978)	EM 280	Comb[5]	1	21	65	260			69g	1.02	1.00 to 1.04			.37
Quadeer et al. (1977b)	EM 210-273[6]	Comb	4	18-36	167-340	700-1229			.70g/d[9]					
Sorensen et al. (1980)	EP 294+EW[3]	Comb	3	20	90-100	350-518	2.6 eggs	.2g						
Venkatramaiah et al. (1986)	EM 280	Comb[5]	2	25-30	107-163	319-589	1.8 eggs	.4g	152g	.66	.59 to .71			
Weyde and Liljedahl (1984)	EM 294[3]	Comb	4[7]	19.4	114.4	445.1	2. eggs	.4g	145g[10]					

[1] EP = egg production, EW = egg weight, EM = egg mass, 280 = part record to 280 days of age, 294 = part record to 294 days of age, 210-273 = recording from 210 to 273 days of age. [2] Comb = combined (Ind, FS, and HS) selection. [3] Mean of period. [4] Mean daily egg mass after AFE. Males were measured for packed semen volume (PSV); in two lines high PSV males were used, while two other lines employed intermediate PSV males. [5] Osborne Index (Osborne, 1957a, 1957b). [6] The period from 210 to 253 days of age was used in the 1st generation of selection. [7] Continuation of the index lines (EP + EW) of Liljedahl and Weyde (1980). [8] Hens housed (or pullets). [9] Reported to be slightly higher (.75g/d) and significant based on 7 generations of selection (Bhagwat and Craig, 1978). [10] Approximated from graphical data.

without direct empirical comparisons the degree of equivalence is far from certain.

Similar to selection for egg production, part-record selection for egg production plus egg weight or egg mass resulted in earlier sexual maturity in 13 of 19 lines (Table 29.16). There was a trend for increased body weight with selection for egg production plus egg weight or egg mass (nine of 12 lines) probably due to the influence of egg size. This is in contrast to the results of unitrait selection for part-record egg production (Table 29.14). Only eight of 14 lines selected for part-record egg production plus egg weight or egg mass showed improvement for short-term rate, and Quadeer et al. (1977b) noted no change in part-record egg production. Weyde and Liljedahl (1984) reported a correlated improvement in egg weight and egg mass for the residual record (42-63 weeks), but not for residual egg numbers or rate of egg production from AFE to 63 weeks. Similarly, Marks (1981) found that residual-record (275-494 days) egg mass improved, but there was no change in residual-record rate of lay when selection for egg mass and packed semen volume in the early part record was practiced. Part-record selection for egg production plus egg weight resulted in no change in fertility, viability, or egg specific gravity (four lines), but hatchability declined and albumen height increased in two of four lines (Kolstad, 1980; Sorensen et al., 1980). The increased albumen height was probably due to increased egg size. Bhagwat and Craig (1978) found that social dominance increased in response to selection for egg mass.

The overall results of the Scandinavian selection and crossbreeding experiment are germane to any discussion of improvement in egg mass, although final evaluation required comparing crosses. Specialized selection, involving selection for egg production in one line (see Table 29.13) and selection for egg size in a second line (methods and population sizes were similar to those used for egg production selection) followed by crossing the specialized lines, resulted in egg mass that was equal to or greater than the egg mass of the crosses of two lines selected for both egg production and egg weight (Kolstad, 1980; Liljedahl and Weyde, 1980; Sorensen et al., 1980). In the 4th generation of the Swedish and Norwegian parts, at least one cross of the specialized lines had superior egg mass, although selection responses for egg mass were greater in the index selected lines in the Swedish part (Liljedahl and Weyde, 1980). This was due to heterosis, because the crosses

Table 29.16. Selection for egg number plus egg weight or egg mass: directional[1] summary of correlated selection responses.

Reference	HDR[2]	AFE	Body weight	
			Housing	Mature
Kolstad (1980)	2=	2-		1+, 1=
Liljedahl and Weyde (1980)	1+, 1=	2-		
Marks (1981)	4+	2=, 2-[3]		4+[3]
Quadeer et al. (1977b)	1+, 3=	4=		
Sorensen et al. (1980)	2+	2-		2+
Venkatramaiah et al. (1986)		4-	2+, 1=, 1-	2+, 2=
Weyde and Liljedahl (1984)		1-		
Total	8+, 6=, 0-	0+, 6=, 13-	2+, 1=, 1-	9+, 3=, 0-

[1] +, increase; =, no change; -, decrease.
[2] Part record - see Table 29.15.
[3] One pair of selected lines was significantly different from the other pair.

of the index lines exhibited negative or no heterosis (Kolstad, 1980; Liljedahl and Weyde, 1980). It is difficult to predict the ultimate outcome because cross performance was changing rapidly generation by generation as genetic differences accumulated between lines. Results from several more generations would have been helpful in resolving this situation.

Too few studies report expected selection responses to draw firm conclusions on O/E responses with regard to selection for either egg production plus egg weight or egg mass. However, in all studies reporting egg mass, egg mass improved, and for those reporting egg production and egg weight, both were improved.

Egg production plus multiple traits. Some of the present knowledge on multiple trait selection is derived from the commercial RRS programs of *H & N Inc.* and *Lohmann Tierzucht GMBH* (von Krosigk et al., 1973; Flock, 1974, 1980, 1988). *H & N Inc.* adopted a RRS program in the 1950s, but this was modified to include a degree of pure line selection at the cost of some heterosis (Flock, 1988; Ameli et al., 1988). In recent years, 87 sires and 870 dams per line were used (Ameli et al., 1988). Selected females were reproduced from an average of 49 sires and 200 dams, and selected males from an average of 25 sires and 68 dams. As expected, there is still much detail about the precise selection procedures used by these companies that remains unknown, but genetic parameters and estimates of genetic progress for several important traits have been published. von Krosigk et al. (1973) reported genetic gains over five years of data from the United States (USA) and seven years of data from Germany for part record and whole record, with expected responses for two years of the USA data. Genetic gains per generation of 1.5 percent (USA) and 1.2 percent (Germany) were recorded for rate of egg production to 305 days, with responses of 1.4 percent (USA) on the complete record (504 days). Viability and egg specific gravity increased. AFE and egg weight remained constant and body weight decreased. O/E genetic response for rate of egg production was 1.38 (USA) and O/E responses for other selected traits ranged from -1.00 to 3.09.

Recent publication of results from 1956-1969 data from *Kimber Farms, Inc.* has also added to the record on multiple-trait selection (Kashyap et al., 1981; Bennett et al., 1981a). Because *Kimber Farms* is no longer an active commercial poultry breeding operation, considerable detail is given on the selection methods. Up to 18 traits were used in an index selection scheme, using strain cross data involving modified sequential selection for breeders of several ages. The hens on which the selection was based were from multiple hatches in three seasons and were tested at 20-40 locations per year. No attempt was made to control management. Bennett et al. (1981a) noted that the overall average selection intensity was 20 percent greater than that intended, but less than intended for some traits such as viability. The correspondence between observed and intended selection intensity changed with breeder age (generation) and period of estimation. Significant genetic gains were reported for whole-record rate, with correlated improvements in total egg production, but gains for pedigreed strain-cross birds were greater than those of commercial crosses. Almost all selected traits experienced positive improvement or no significant change. Observed genetic changes were larger than predicted for rate (O/E: 1.14 linear and 1.77 quadratic estimates) and egg production (O/E: 1.41 linear and 2.10 quadratic estimates). However, these estimates of genetic progress depend heavily on the reliability of the repeat mating genetic control (see Chapter 38) used in this study, which had very high mortality. Mortality is a very complex trait; resistance to each pathogen should be considered a separate trait. With the high levels of mortality in the study, it is doubtful if this genetic control was as useful as it would have been had mortality been lower.

A 30-year selection program at the Animal Research Centre of Agriculture Canada, Ottawa provided considerable information on several aspects of multiple-trait selection (Gowe et al., 1973; Gowe, 1974, 1977; McAllister, 1977; Gowe and Fairfull, 1980, 1982a, 1982b, 1984, 1985, 1986; Fairfull and Gowe, 1980; Fairfull et al., 1983, 1987). A consistent multivariate culling levels scheme was employed in the selection of all strains. The selection practiced for females is summarized in Table 29.17. When selecting males, the full-sib and half-sib family information of females was used. All selected males and over 90 percent of selected females came from half-sib families that ranked above the population mean. Over 75 percent of the males came from the top 25 percent of the half-sib families. Despite the emphasis on half-sib families, a minimum of ten half-sib families contributed sires each generation to limit the rate of inbreeding. From the time of separating each base into two selected lines (1969 or 1971) until 1980, each selected line used 28 sires and 224 dams with about 1100 hens housed per strain. In earlier years, the populations were larger (see Gowe and Fairfull, 1985). The control strains are described in Chapter 38.

Retrospective analysis of the data from 1971 to 1975 (McAllister, 1977) showed that the emphasis (mean selection coefficients = average of male and female selection) on traits was consistent in the two sets of strains: .21 for HHP1 in Strains 3, 4 and 8 vs. .21 for HDR1 in Strains 1, 2 and 9. The emphasis was .09 for egg weight in both sets of strains. The relative emphasis for traits was 15.5 : 6.3 : 3.4 : 1 for HHP1 or HDR1, egg weight, specific gravity, and fertility respectively. Very little pressure was required to maintain fertility, hatchability, and brooding and rearing viability at high levels in this selection scheme (across strains and sources of information, very few selection coefficients were significant). The emphasis for Haugh units and blood spot incidence was similar to that for specific gravity, but slightly greater than that for laying viability with brooding and rearing viability, and hatchability receiving a little more emphasis than fertility. The relative emphasis on source of information was 3.3 : 1.8 : 1 in HHP1 or HDR1, and 1.5 : 1.5 : 1 in egg weight for half-sib family, full-sib family, and individual information, respectively. Because these 'index in retrospect' calculations were based on assumptions of genetic additivity, and because there were important nonadditive effects for egg production (see **Nonadditive inheritance**), the index in retrospect estimates of relative emphasis on source of information for egg production will be biased by the amount of nonadditivity. The mean correlation between assessed and predicted overall merit was .75, ranging from .74 to .77 among strains.

All selected strains had highly significant genetic gains for the egg production trait selected (Figures 29.1 and 29.2), egg weight (see Chapter 38), egg specific gravity and Haugh units (see Chapter 38) over the periods that they were selected (Table 29.17; Gowe and Fairfull, 1985). In the few years of selection for reduced 265-day body weight, there were significant correlated reductions in mature body weight (365 days), but the direct response for 265-day body weight was not significant. As traits were added to the selection program over the generations, selection pressures mainly for egg production and egg weight were reduced, and as a consequence, selection responses were reduced also (Gowe and Fairfull, 1985). There were some small desirable changes in brooding and rearing viability, and in hatchability for some strains, but the generally good performance for these traits resulted mainly from maintaining the high performance of the original lines plus improvements in the environment. Despite the increasing levels of inbreeding, all selected strains remained at previous high levels of fertility and viability with any major improvement being environmental, for example, the increase in fertility with the introduction of artificial insemination or the decrease in Marek's disease mortality with the introduction of Marek's disease vaccine. The strains selected for HHP1 had greater gains for

Table 29.17. Agriculture Canada multiple-trait selection study: selected trait, year of initiation of selection, approximate selection emphasis within trait, and estimates of genetic gains and correlated responses per generation for hen-housed egg production to 273 days (HHP1: strains 3, 4 and 8) and hen-day rate from AFE to 273 days (HDR1: strains 1, 2 and 9) for selected strains from 1971 to 1980 calculated as the deviation from the mean of unselected control strains 5 and 7.

	Initial		Approximate selection emphasis within trait			
Selected trait	year	Strains[1]	HS[2]	FS[2]	Indiv.	Pedigree
Hen-housed egg production to 273 d	1950	3, 4[3], 8[4]	++++	++	+	
Hen-day rate from AFE to 273 d	1969	1[5], 2, 9[4]	++++	++	+	
Brooding and rearing viability[6]	1950	All	++	+		
Laying house viability to 273 d	1950	All	+	+	++	
Fertility	1952	All			+	++
Hatchability	1952	All				++
Egg weight at 240 d	1952[7]	All	+	+	+++	
Egg specific gravity at 240 d	1960[8]	All	+	++	++++	
Haugh units at 240 d	1960[8]	All	+	+	++++	
Blood spot incidence at 240 d	1960[8]	All	++	++	++	
Egg shape at 240 d	1960[8]	All	+	+	++	
Body weight at 265 d	1975	All	+	++	++	

Genetic gain per generation	Selected for — HHP1	HDR1
SELECTED TRAIT		
Hen-housed egg production to 273 d, no.	1.24**	
Hen-day rate from AFE to 273 d, %		.44**
Fertility, %	.11	.10
Hatchability, %	.18	.31*
Brooding and rearing viability[6], %	.13	.23*
Laying house viability to 273 d, %	.01	.01
Egg weight at 240 d, g	.15**	.16**
Egg specific gravity at 240 d	.32*	.38**
Haugh units at 240 d	.36**	.56**
Blood spot incidence at 240 d, %	-.03	-.04
Egg shape at 240 days	.00	.00
Body weight at 265 d, g	-4.	-5.
CORRELATED TRAIT		
Hen-housed egg production to 273 d, no.		.16
Hen-housed egg production to 497 d, no.	1.61*	2.08**
Hen-day rate from AFE to 273 d, %	.16	
Hen-day rate from 274 to 385 d, %	.12	.72**
Hen-day rate from 386 to 497 d, %	.11	.98**
Laying house viability to 497 d, %	-.13	-.11
Age at first egg, days	-1.14**	.39
Egg weight at 450 d, g	.10	.06
Egg specific gravity at 450 d	.22	.34*
Mature body weight, g	-28.**	-26.**

[1] Three genetic bases were used: an Agriculture Canada gene pool present prior to 1950 (unselected control strain 5, and selected strains 1 and 3), a synthetic of Canadian R.O.P. stocks started in 1951 (selected strains 2 and 4), and a synthetic of 4 North American commercial stocks started in 1959 (unselected control strain 7, and selected strains 8 and 9). [2] HS = half-sib, FS = full-sib family information. [3] Initial year 1951. [4] Initial year 1969. [5] Initial year 1971. [6] Selected as separate traits. [7] Pedigree selection to 1959, family plus individual selection from 1960 to 1980. Between 1973 to 1976 depending on the egg size of the strain, selection was redirected to maintaining rather than further improving egg weight. [8] From 1969 for strains 2, 8 and 9, from 1971 for strain 1. * P>0.05. **P>0.01.

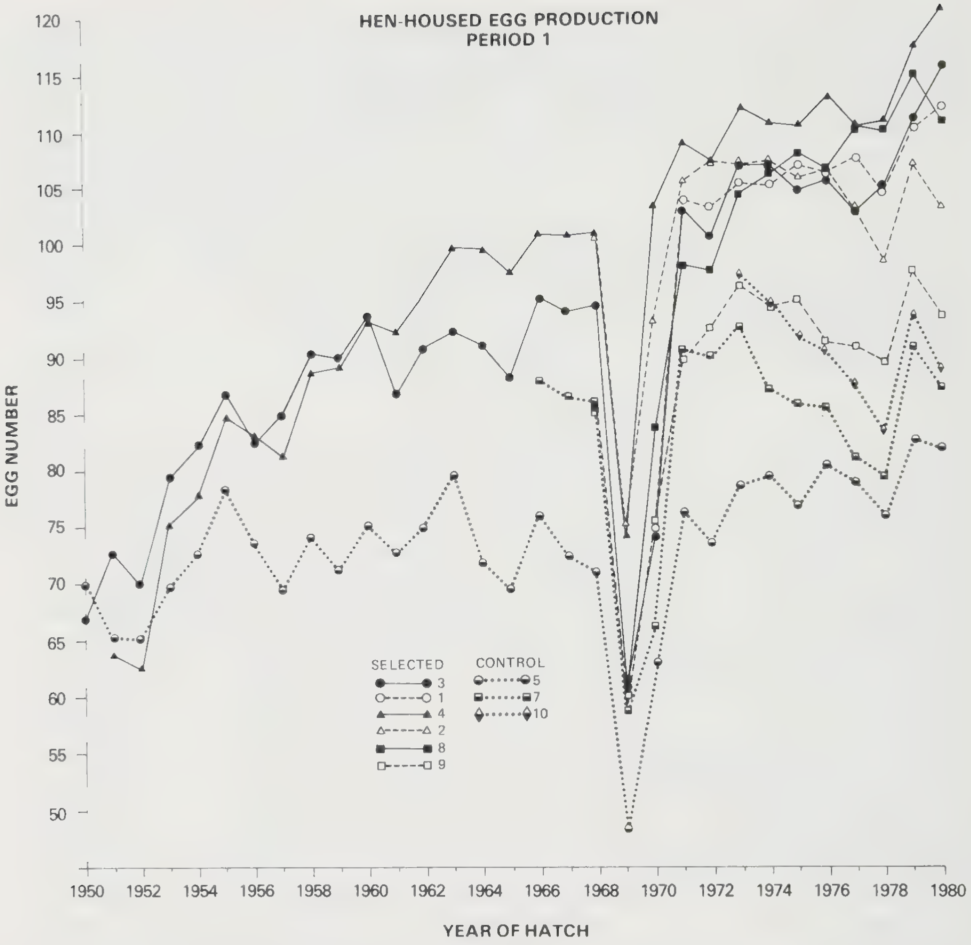

Figure 29.1. Hen-housed egg production to 273 days for the selected (1, 3, 2, 4, 9 and 8) and unselected control (5, 7 and 10) strains.

HHP1 than the HDR1-selected strains and vice versa.

The HHP1 selected strains all had increasingly earlier AFE which accounted for most of the improvement in HHP1 (Table 29.17). From 1971 to 1980, the HHP1-selected strains showed no significant improvement for HDR1 (despite improvements over the first few generations of selection in the long-term selected strains), HDR2 or HDR3 (Figure 29.3), and thus, the improvement in HHP5 (Figure 29.4) was mainly due to earlier AFE. Two of the three HDR1-selected strains (strains 2 and 9) had significantly later sexual maturity (see Gowe and Fairfull, 1980, 1986), although the overall later AFE was not significant (strain 1 had a nonsignificant trend for earlier AFE), so that despite the improvement in HDR1, HHP1 did not change significantly. HDR2 and HDR3 improved in the HDR1 selected strains, but improvements in HHP5 were reduced due to later sexual maturity in two of the three strains.

These reports on multiple-trait selection showed that egg production was improved

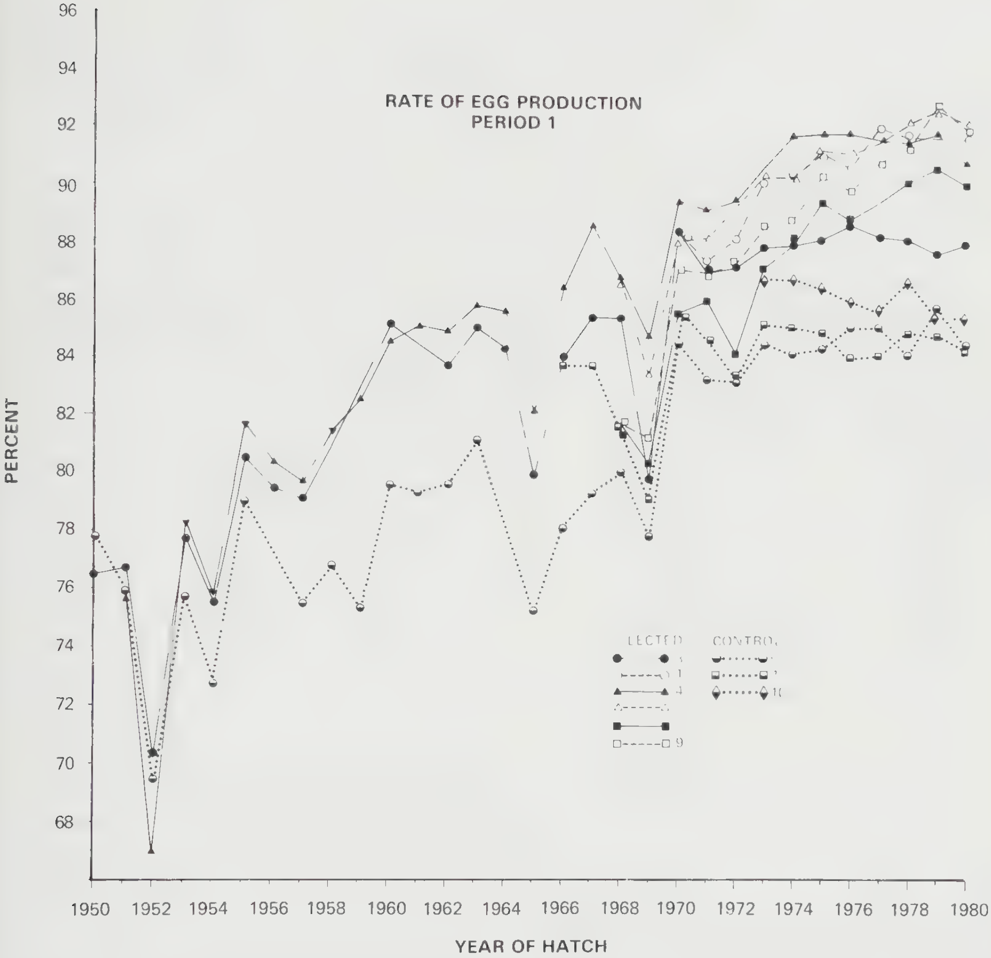

Figure 29.2. Survivor hen-day rate of egg production from first egg to 273 days for the selected (1, 3, 2, 4, 9 and 8) and unselected control (5, 7 and 10) strains.

while several other important traits were improved or maintained at high performance levels despite the fact that desirable performance in most of these traits was antagonistic to high egg production. Also, positive genetic gains were realized in each of the improvement schemes employed.

Effects of selection.

Changes in genetic parameters. Selection reduces genetic variation as expected (see the **Heritabilities** section). Most of this reduction usually occurs in the first few generations (see Yamada et al., 1958, and Gowe and Fairfull, 1985). The question remains whether the residual variation dwindles slowly to insignificance and disappears.

The experimental evidence indicates that a substantial level of genetic variation is

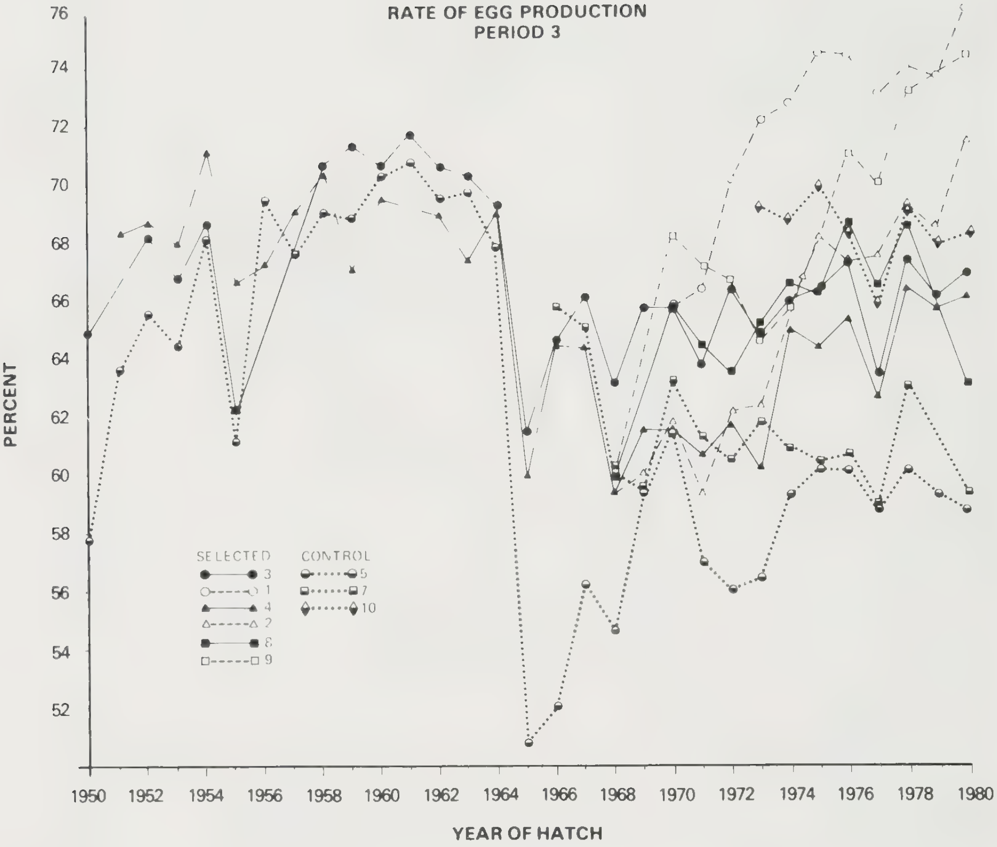

Figure 29.3. Survivor hen-day rate of egg production from 386 to 497 days for the selected (1, 3, 2, 4, 9 and 8) and unselected control (5, 7 and 10) strains.

residual in populations. Over ten generations with a limited population of White Leghorns (4-20 sires, 45-132 dams, 255-877 hens housed per generation), Yamada et al. (1958) selected for part-record rate of lay as the primary trait along with viability, hatchability, and egg size. Although variability seemed to decrease, there was considerable genetic variation at the end of the study. Given the population structure and size, it is doubtful that heritability changed from the third to tenth generations. Friars et al. (1962) selected for juvenile body weight and breast conformation, adult body weight, egg production, and egg weight over nine generations (5-18 sires, 32-125 dams, 106-543 adult progeny) in a population synthesized from several breeds. They found no change in the heritability of egg production. Goher et al. (1978a) reported no significant change in sire variance components during 11 generations of selection (see Table 29.13 for details of selection). In the California selection study in which Lerner (1958) used White Leghorns, the heritability of egg production traits remained constant over about 15 generations of selection for hen-housed egg production, egg weight and other characters. Gowe and Fairfull (1985) selected

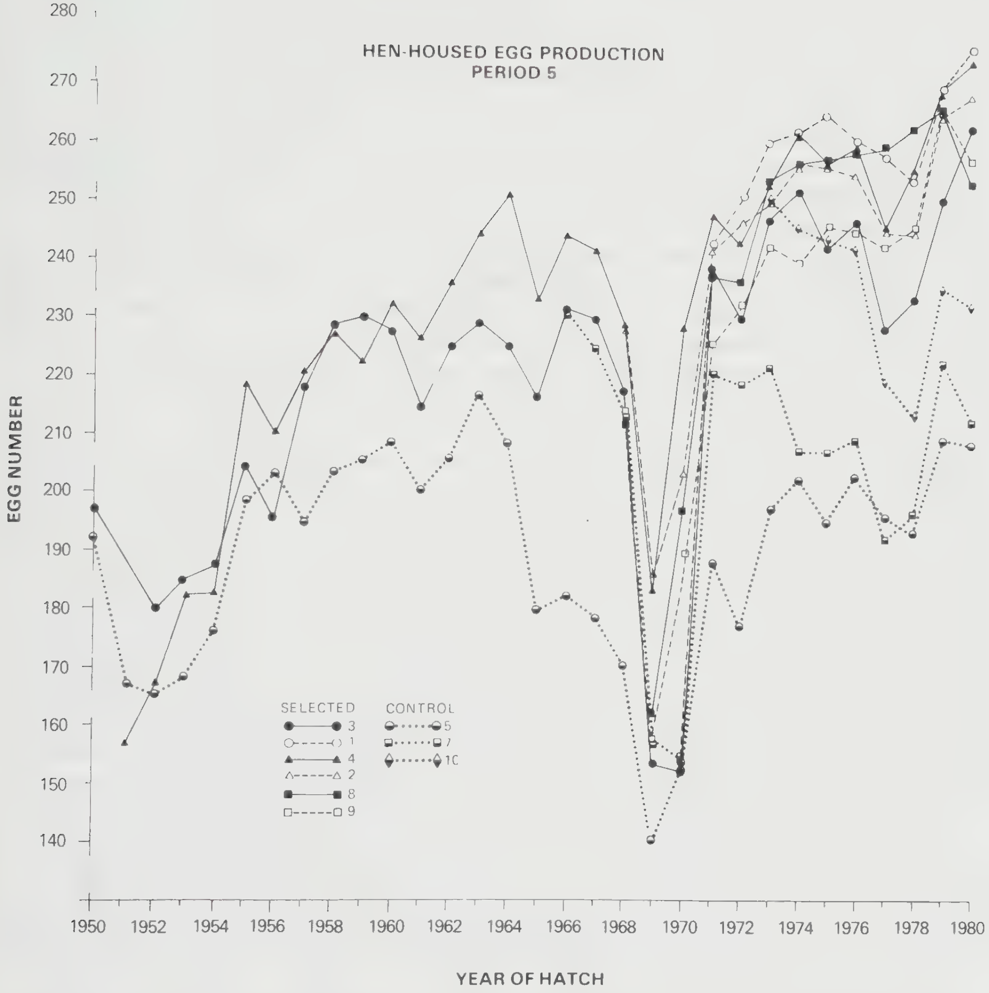

Figure 29.4. Hen-housed egg production to 497 days for the selected (1, 3, 2, 4, 9 and 8) and unselected control (5, 7 and 10) strains.

for hen-housed egg production, viability, fertility, hatchability, egg weight, and other economic traits in two White Leghorn genetic bases for 29 and 30 generations, respectively. No significant decrease in the additive genetic variation or heritability of egg production characters was found. However, the genetic variation in the unselected base population was higher than that of the selected strains. Thus, the evidence is fairly convincing that even with fairly small populations, providing selection intensity is reasonable, residual genetic variation remains relatively constant and selection response continues.

That genetic variation in selected populations remains at a substantial fraction of that estimated in unselected populations would not have been accepted as orthodox quantitative genetics theory a short time ago. However, empirical results from selection studies like those discussed above (and from other species), and new discoveries about DNA and the facility with which it can change, refute this orthodox simplistic theory. Frankham (1980, 1983) showed that mutations make a much greater contribution to genetic variation in selected lines than was previously thought possible and this has stimulated theoreticians to revise their views on genetic variation.

Genetic relationships among important traits can be changed by selection also, although the facts suggest that there is limited predictability. Evidence of changes in r_g values is fairly common. Disparities between the r_g values of control and selected lines in Tables 29.4 to 29.12 reflect this, although by itself this cannot be considered proof. Ibe et al. (1982) reported significant curvilinear trends in correlated responses to selection for whole-record egg numbers and rate, and egg and body weight. Morris (1963) noted changes in r_g of early and late egg production resulting from selection. Liljedahl and Weyde (1980) reported that the r_g between part-record egg production and egg weight became negative as a result of part-record selection for egg production. Similarly, Poggenpoel and Erasmus (1978) noted that realized correlated responses in egg weight resulting from selection for part-record egg production were several times larger than expected. Gowe and Fairfull (1985) published differences between the r_g values of control and selected lines. There were differences also between the genetic correlations of the two selected lines reported in Gowe and Fairfull (1984) that may be significant. However, the reader should not lose sight of the fact that many r_g values remain relatively constant, either without much change or after an initial change (Gowe and Fairfull, 1985).

The simplest mechanism for changes in r_g values is the dissociation of linkages. Linkages dissipate at various rates depending upon their closeness and other biological phenomena enhancing or inhibiting crossovers, so that such changes could potentially occur over a significant time. Changes in pleiotropic relationships might occur also and these might be subtly affected by shifts in gene frequencies, changing the dynamics of a complex of biochemical pathways. Interested readers are directed to Sheridan and Barker (1974) for a discussion of two theories of shifts in genetic correlations involving pleiotropic genes.

Inbreeding. By definition effective selection involves inbreeding. Selection results in the reproduction of individuals with above average genetic merit, which in turn causes a higher degree of relationship among their progenies than would have resulted with no selection and only true random mating. The degree of inbreeding is dependent upon population size, selection intensity, and any restrictions placed upon the use of related individuals as breeders. A frequent restriction by breeders is to set a minimum number of sire or dam families that must contribute breeders to produce the next generation of progeny.

There is considerable literature to show that rapid inbreeding results in a general loss of reproductive (fitness) and productive performance of a chicken population (see Chapter 39). With rapid inbreeding, inbreeding coefficients calculated from the pedigree more closely approximate the actual degree of inbreeding than otherwise; inbreeding as calculated from pedigrees overestimates gene fixation (Nordskog and Hardiman, 1980). The traits affected and degree of effects due to inbreeding in chickens vary from population to population (Ibe et al., 1983).

Ibe et al. (1983) used full-sib matings to reproduce two White Leghorn lines (64 males and 168 females each) over 19 generations. One line was selected for part-record rate of lay. Compared to a control, both lines experienced significant declines in egg production and there was no real difference between the selected and unselected lines. Nordskog and Hardiman (1980) selected six White Leghorn lines for rate of lay, egg weight to body weight ratio, or egg mass to body weight ratio, using eight sires per line mated to two full-sibs, two half-sibs, and four non-sibs, over four or seven generations. The average rate of inbreeding was 2.4 percent per generation. With an effective population size of 20, they estimated that inbreeding depression would reduce selection response for rate of lay by 25 percent (53 percent for hatchability and 41 percent for viability). Obviously, rapid inbreeding is detrimental and may not be amenable to alleviation by selection. Also, hatchability is very sensitive to inbreeding, and as such is a fairly reliable indicator of inbreeding.

In the California selection experiment (Lerner, 1958), the rate of inbreeding was just under one percent per generation. From indirect evidence, Lerner (1958) speculated that the expected gain was reduced by 10-20 percent due to inbreeding. However, hatchability and fitness (number of offspring surviving to eight months of age) improved, indicating that the effects of inbreeding on egg production may have been overestimated.

Gowe's selected lines were inbred at a rate of about .7 percent per generation (Gowe and Fairfull, 1980, 1982b). The degree of inbreeding, that varied from 20-25 percent in the long-term (29 or 30 generations) selected lines, apparently did not have a large effect because egg production traits, fertility, hatchability, and viability improved. In this study, selection for fertility and hatchability was on a pedigree basis, and small fluctuations in fertility and hatchability were largely ignored. Only males and females from parents with very low values were culled. Hence, selection for the fitness traits (fertility and hatchability) was slight, and yet no discernible inbreeding depression was evident (compare Ibe et al., 1983). Also, quantitative genetics theory states that genetic variation should be reduced proportionally to inbreeding, which was not the case in this study (Gowe and Fairfull, 1985). However, care was taken to prevent rapid inbreeding in these moderately large breeding populations as pointed out earlier.

Although inbreeding depresses egg production traits and reduces selection response, its effects are most pronounced when there is rapid inbreeding and little opportunity to alleviate adverse effects by selection. With rapid inbreeding, the actual degree of genetic fixation is closer to the calculated level than with slow inbreeding. When the rate of inbreeding is low, theoretical inbreeding coefficients calculated from the pedigrees probably overestimate gene fixation and the consequent detrimental effects on selection response. Also, mild selection can eliminate undesirable recessive genes uncovered by inbreeding. Thus, the results of selection studies with very small populations or very intense selection with rapid inbreeding cannot be transferred to large populations with low inbreeding.

Selection limits. Selection limits (or selection plateaus) are extreme levels of production which cannot be surpassed. Theoretically, selection limits can result from the exhaustion of genetic variation, biological or physiological limits, loss of fitness, or antagonistic genetic relationships among selected traits. Overdominance can also limit selection response (see Al-Murrani, 1974), but this is unlikely in the case of egg production. Although upper limits to selection have been the subject of extensive discussion and theoretical consideration, plateaus have no empirical support in practical or industrial selection in egg (or meat) stocks of poultry despite premature assessments to the contrary in past decades (Dickerson, 1955; Clayton, 1972; Nordskog, 1975). Moreover, experimental evidence of plateaus even in relatively small populations is not convincing. This does not mean that selection limits cannot be reached (see Clayton, 1972), but the large populations used by applied breeders make this unlikely until higher rates of egg production have been achieved.

In commercial stocks, there is no evidence of upper limits to selection as yet (Hunton, 1984; Flock, 1988; McMillan et al., 1989a). There is no reason to expect a reduction in progress unless selection pressure for egg production is reduced. There are some reasons to expect that the rate of genetic progress could be improved by clearer selection goals, better trait definition, more efficient selection methods, and by optimizing the part-record selection period among other factors. Commercially selected populations are large, and selection pressures are well beyond those within the reach of most academic studies; these populations are unlikely to plateau until they approach production rates of an egg per day.

The early experimental evidence of selection limits is not entirely credible. The evidence for a selection limit presented by Yamada et al. (1958) depends mostly on a single low point in the last generation. Also, the generation means preceding it could be high which would tend to distort response. The studies of Ibe et al. (1982) and Goher et al. (1978a, 1978b) have not clarified the plateau situation in small populations.

The exhaustion of genetic variation is unlikely to be the cause of a plateau for egg production in chickens (see *Changes in genetic parameters*). Biological or physiological limits have resulted in selection plateaus (lower limits) in other species, and it is probable that such an upper limit exists for egg production in chickens. Although it should not be a problem in the near future, the biological limit of one egg per day may eventually result in a selection plateau for egg production. Even this may be postponed by modifying the environment (see **Lighting**) as discussed by Foster (1985) and Yoo et al. (1984, 1986).

In the selection of layers, the important components of fitness are usually considered in the selection scheme (i.e. fertility, hatchability, viability, egg size, etc.), and egg production itself is a component of fitness. The studies of Gowe and Fairfull (1985, 1986) and Lerner (1958) demonstrate that fitness can be maintained or increased as egg production is improved. Thus, a loss of fitness need not be the cause of selection plateaus for egg production.

Simultaneous selection for several antagonistic traits could minimize selection progress and theoretically stop it (a balance between natural and artificial selection could be seen as a corollary). This could possibly explain some differences in responses among selected lines. However, selection in chicken egg stocks presently includes up to 14 traits (and sometimes more) including egg production, egg weight, body weight, sexual maturity, egg shell strength and feed conversion, among which there are several antagonistic relationships, and yet substantial progress is achieved (see Gowe and Fairfull, 1984, 1985, 1986; Hunton, 1984; Flock, 1988; McMillan et al., 1989a). If enough traits were included in the selection rule, progress could cease due to the reduction in selection pressure on traits already included (Gowe and Fairfull, 1985).

Of all the possible causes of selection plateaus for egg production in chickens, exhaustion of genetic variation and loss of fitness do not appear to be likely causes in large populations with reasonable selection programs. However, it seems probable that a biological limit exists with regard to selection for high egg production although this limit has not yet been reached.

Part-record selection. Almost all of the selection studies described in preceding sections of this chapter used part-record selection. The important results of part-record selection are listed there with the corresponding literature.

The following discussion is based upon the assumption that the selection objective is improved whole-record egg production in a multiple-trait selection scheme. For objectives other than the maximal improvement of whole-record egg production, an egg production trait or part-record of selection other than that suggested here may best suit that objective.

There are a number of important empirical findings with respect to part-record selection. They were found because long-term selection studies were conducted in some cases and some researchers collected records on the complete laying cycle. Part-record selection has been discussed in detail by Bohren (1970) and Bohren et al. (1970).

Selection for HHP on a part record. Selection for part-record HHP acts to improve egg production mainly by decreasing AFE. Rate of egg production may improve for some parts of the laying cycle early in the selection program, although no improvement was found in some studies. However, after a number of generations in long-term studies, improvement in egg numbers in the residual record stopped and the correlation between the part and residual record decreased, and in some cases it became antagonistic. Use of a longer part record of selection would likely have reduced this effect resulting in less indirect pressure on AFE and more on HDR.

Selection for HDR on a part record. There are some differences between one-trait and two-trait selection studies, and those using multiple (three or more) traits. In the former, most lines selected for part-record HDR from AFE had earlier or unchanged AFE. In multiple-trait selection studies where AFE was not under direct selection, the tendency was for later AFE or no change; in the lines with increasingly later maturity, egg numbers in the part record changed very little although part-record and residual-record rate were substantially improved.

Selection studies have not answered a number of important questions with regard to part-record selection. Two of these are: what is the optimum record for selection to improve whole-record egg production; and, what is the optimum egg production trait (or combination of traits) to use in selection?

Part-record length. The optimum length of a part record as a selection criterion has been studied by statistical examination of records of various lengths. However, most such studies have contributed little to this particular objective for a variety of reasons (Dhaliwal and Acharya, 1972; Chaudhary et al., 1975; Lowe and Garwood, 1980; Foster, 1981b; Hanafi and El-Labban, 1984; Singh and Mohanty, 1985). The studies of Flock (1977, 1980) focused on the optimum length of a part record as a selection criterion. He reported that a test of 32 weeks in the laying house should be close to the optimum. With this criterion, the generation interval would be approximately 15 months. McMillan et al. (1989b) approached the subject from a different perspective. They fitted postpeak weekly egg production to various part records ranging from 16-40 weeks after AFE to predict the residual record, and thus to predict full-record egg production of individuals. There was a point of inflection in the area of 28-36 weeks of test, which indicates that the optimal length was in this region. Also, the correlation of the predicted residual and actual residual records averaged more than ten percent higher than that of the actual part record and the actual residual record.

Egg production trait. Hen-housed egg number does not seem to be the best measure of egg production for part-record selection because it puts a lot of pressure on AFE and viability, and little on rate, especially rate in the residual record. In newly synthesized populations that are very late maturing, this measure might be used successfully for the first few generations. Rate of egg production is not entirely free of problems associated with indirect selection pressure on AFE. In multiple-trait selection, the direction of change in AFE was undesirable. AFE must be selected for independently in part-record rate selection programs and viability must also be selected for independently, but this is the most effective way to deal with all three traits. There is no final definitive answer in the choice of an egg production trait for selection. As the length of the egg production record used in selection approaches the whole record, the less meaning any difference will have.

However, with the limited evidence available, predicted full-record egg production based on a part record of about 32 weeks would seem to be the best egg production trait to use in a multiple-trait selection program. However, the selection program should be carefully monitored to ensure that there is continuous progress. The second choice would be HDR from AFE to 32 weeks, with selection for AFE and viability as needed.

Nonadditive inheritance. Nonadditive genetic effects are very important for egg production in chickens (Sheridan and Randall, 1977; Sheridan, 1980; Bennett et al., 1981b; Fairfull et al., 1983, 1987; Gowe and Fairfull, 1982a). Temporary epistatic and maternal effects distort the relative value of family means, especially full-sib families, in selection. Bennett et al. (1981b) concluded: "Temporary epistatic and maternal responses can explain overestimation of additive genetic response from offspring-parent regression or from replicated single-generation selection and apparent superiority of mass selection over family or combined selection." The results of studies comparing selection for egg production based on individual, family, or combined selection (Kinney et al., 1970; Garwood and Lowe, 1979), especially if they are single generation studies (Garwood et al., 1980; Garwood and Lowe, 1981), are biased by temporary epistatic and maternal effects in addition to the confounding effects of inbreeding, population size and structure (where present). Procedures such as index selection do not normally take the effects of temporary epistasis and maternal effects into account in weighting the relative value of individual, full-sib, and half-sib information. In such cases, index selection overvalues full-sib information. However, if the parameters are known and considered in construction of the index, temporary epistasis and maternal effects can be treated as common environmental effects in index selection. However, the problem in practice is that the magnitude must be known. This subtlety is very important for egg production, a trait that is heavily influenced by nonadditive gene action (Fairfull et al., 1987), but has little influence when selecting for traits such as body weight.

Selection can only sustain improvement due to additive gene action. Linkage-related epistasis will dissipate as crossing over breaks up desirable gene combinations. Birds with congenital disease infections will tend to be eliminated from the population as selection progresses. Additive x additive effects will have only short-term importance. Only mating plans such as line crossing, specialized sire and dam lines, or recurrent (or RRS) selection can be expected to consistently utilize nonadditive gene action.

Multiple Trait Selection: Methods And Criteria

Traits. Many traits are important in the selection of laying hens: egg production, fertility, hatchability, viability during the brooding, rearing and laying periods, blood spots, meat spots, age at first egg, egg weight, body weight, egg shell strength, Haugh units, feed conversion, egg shape, egg shell texture and egg shell color. The relative importance of traits varies among market areas of the world. Not all traits are economically important in all markets particularly in the long-term planning of breeders.

Layer traits fall into several classes that influence the manner in which they are treated and the results of the selection practiced. Some of these categories are fitness traits (such as fertility, hatchability, viability, egg production, and egg size), all-or-none traits (such as blood spots, meat spots, and viability), discontinuous traits (such as age at first egg, egg specific gravity, and egg production), and continuous traits (such as egg weight, body weight, egg shell weight, most egg shell strength measurements, Haugh units, feed conversion, egg shape, egg shell texture, and rate of egg production).

Many of these traits, such as the feed conversion and egg shape ratios, are not normally distributed. Ratios are generally capricious traits upon which to practice selection. For example, hens with very low feed consumption resulting from subclinical disease infections may appear to have excellent feed conversion, even when other performance characteristics are well below par. Also, it is critical to have birds that have sufficient appetite to withstand periods of stress. These may not be the hens with the very best feed conversion. Fitness traits generally have low or very low h^2 values. The all-or-none traits often have incidences that are very low. Frequently, after several generations of selection, the incidence is so low that only selection against undesirable extremes is practical. The trait cannot be excluded from the selection rule because it cannot be allowed to increase, but it would not be cost effective to put much selection pressure on the trait.

Trait definition plays an important practical role in layer selection. Trait definition includes exactly how the trait will be measured, how any calculations will be done, a clear definition of any exclusions or edits, when the character will be measured, and the length of the measurement period. The following is not intended to be a comprehensive discussion, but simply to illustrate some problems in trait definition. For both egg number and hen-day rate, many of the important issues of trait definition are discussed in **Part-Record Selection**.

Trait definition is also important for traits other than egg production. Many traits change with age in a nonlinear fashion. Egg weight increases with age, while egg specific gravity decreases with age. The age at which traits are measured will frequently affect genetic parameters and the efficiency of using the information. Egg shell strength measurements should probably be taken as late in the part-record period before selection as is possible, because the r_g values of egg shell strength measurements with proportion of intact eggs late in the laying cycle increase with age (Grunder et al., 1989). The correlation with late breakage is important because early breakage is less important and more nearly random. Also, for egg shell strength, the traits used to assess it for selection are indirect and there are many alternatives (egg specific gravity, compression fracture strength, deformation, egg shell weight, egg shell thickness, egg shell weight per unit area, etc.). These alternatives are not equal with respect to effectiveness in predicting breakage and cost of measurement. At the present state of knowledge, egg specific gravity measured by flotation would seem to be one of the best alternatives (Grunder et al., 1989; Strong, 1988).

Methods. There are three classes of selection methods, each with several variants: independent culling levels, index selection, and tandem selection. *Tandem selection* refers to the practice of selecting one trait for one or more generations, followed by selection of another trait for one or more generations, with subsequent corresponding selection for the remaining important traits. With tandem selection, the number of generations of selection might be proportional to the importance of the trait. Tandem selection is inferior to the other selection methods (Hazel and Lush, 1942), although it may be useful in specific instances. *Independent culling levels* is the practice of setting culling levels (a standard below which animals are not considered as candidates for breeding) that are applied independently for each selected trait. *Index selection* uses a linear selection rule derived by defining an idealized aggregate genotype whose phenotype is equated to its genetic expectation, using the information from all relevant sources to derive a single relative value that can be ranked for selection.

Sequential selection or multiple stage selection is the application of selection criteria in series over time, often before breeding age, by whatever selection method is being used. A clear example of sequential selection occurs in broiler female (dam) line selection. Growth rate (or size at a constant age) and conformation are very important traits and assessment can be done at a much earlier age than female reproductive traits. The reduction in population size by selecting for growth rate at broiler age results in a more economical laying test without much reduction in selection efficiency, and may even enhance it because a much larger population usually can be tested for growth than can possibly be tested for egg production.

Selection using independent culling levels (ICL) is usually viewed as simple mass selection. In fact, ICL selection can be a complex and versatile method using information from multiple sources. In multiple trait selection, culling levels can be adjusted empirically to achieve the desired goal. ICL selection can be varied to suit the trait. Full-sib and half-sib family information can be combined with individual information in ICL selection. Culling levels can be varied according to a specific set of criteria, for example, with family ranks or with family size or within-family variation or with some combination of these. For many characters, ICL selection may be more effective and valid than index selection (Frankham et al., 1989), as Gowe and Fairfull (1982b) suggested. ICL selection does not require the assumptions of index selection and is very effective in practice. This is particularly true if r_g or economic weights are poorly known, and in selection of traits controlled by very few loci, or in culling undesirable recessives (mutations) with very low incidence. However, ICL selection cannot fully utilize information from other traits and cannot easily use information from relatives outside of the full-sib and half-sib families.

Index selection uses a linear selection rule (Smith, 1936; Hazel, 1943) that combines all relevant information (including family and individual phenotypic values, genetic and phenotypic variances, and covariances with relative economic weights) into a single value that is ranked for selection preference. Some nonlinear indices have been derived (e.g. Wilton et al., 1968) for specific types of nonlinearity. Although index selection is theoretically best, index selection has numerous problems in practice. Bell (1982a) pointed out that many experimental comparisons using indices were flawed due to parameter estimation errors. Genetic parameters are assumed to be known and unchanging in index selection. However, errors in r_g estimates can be grossly misleading and they are often poorly estimated, especially those with traits that are lowly heritable or have incidences approaching zero or 100 percent. There is no satisfactory and generally accepted method to derive relative economic weights. Relative economic weights are a linear description of a number of complex nonlinear functions in layer selection, and this is unacceptable to many researchers and industrial geneticists. When several traits are selected simultaneously, one or a few important traits tend to dominate the index. There is no practical method of including some traits, such as single gene characters with deleterious phenotypes, in an index.

Best Linear Unbiased Prediction (BLUP) is a set of methods that is equivalent to index selection under some circumstances, but with significant improvements in several areas including statistical methodology (Henderson, 1984). BLUP uses the additive genetic relationships among all animals considered, and thereby improves genetic assessment. When used with a profit function, economic evaluation is much more acceptable. Genetic and economic appraisal are separated, so that they can be checked and evaluated independently. Economic assessment need not be linear, and it can be as complex as is necessary. Application is still very dependent upon accurate r_g values and an accurate economic algorithm.

While it may be acceptable to use BLUP or an index to reduce the complexity of practical selection problems (for example, when there is too much information), some traits cannot be safely or efficiently included at present. In some cases, this is simply because the incidence is too low. In others, the assumptions implicit in the method, such as polygenic control with each gene exerting a small additive effect on the character, may be violated. In layers, fertility may be a good example of this. Fertility has very low heritability, but a few individuals in a large group of matings may have very low fertility, and frequently among their progeny an unusually high number of individuals with very low fertility may occur. This may be due to defective genes or possibly a deletion or a duplication. Such situations are not handled efficiently by index selection or BLUP. However, culling the few individuals and families with extremely low fertility in every generation, and ignoring the normal moderate variation in fertility, works extremely well. Such situations may exist for other characters in many species (see Frankham et al., 1989). At present, the most practical selection method may be to use BLUP or an index for most traits, but to use some form of independent culling levels selection for fertility, hatchability, viability, and blood and meat spot incidence.

In poultry, there are two added selection problems. First, the commercial egg production hen is a strain or breed cross. Within-line selection (WLS) of the parental pure lines maximizes the performance of these parental lines, but does not optimize the use of nonadditive genetic variation. As a result, some layer selection schemes have utilized reciprocal recurrent selection (RRS). RRS is a form of family selection that uses data from reciprocal crosses to select pure line parents. RRS does not maximize performance of the parental pure lines. Some aspects of WLS and RRS are discussed in Chapter 37 and in Saadeh et al. (1968), Calhoon and Bohren (1974), and Bell (1982b). Secondly, in layers, pure strain hens are evaluated for egg production with one hen per cage, but commercial layers are kept several to a cage at a range of stocking rates. Muir (1985; 1988, personal communication) has shown that selection in single-bird cages may not result in as efficient selection for performance at very high stocking rates as selection utilizing group performance data.

If information from pedigreed strain cross tests in colony cages, or from testing pedigreed pure strains in colony cages, were included as correlated traits in a BLUP or index analysis, some of the inadequacies of present technologies might be alleviated. Further research is necessary to determine if the increased efficiency is cost effective. However, both Arthur (1986) and Flock (1988) have suggested that most breeders are probably using a combination of pureline and crossline testing.

Population size and structure. The breeding structure of a population is an important ingredient for accurate genetic assessment. In poultry, the most common (almost exclusive) breeding structure is hierarchical with one male (sire) being mated to several females (dams).

Each dam's genotype is evaluated with only one genotype (one sire), so that genetic assessment is relatively poor. In estimation of the dam family mean, precision depends upon the number of progeny tested per dam, and environmental bias depends upon the representation of the progeny across various potential macroenvironments (i.e. brooding and rearing or adult: cage or pen, tier, battery, wing, house; disease exposure, etc.). Each sire's genotype is assessed with several female genotypes, so that genetic assessment is comparatively good. The estimation of the sire family mean is generally more accurate and precise than that of the dam family mean because of the greater number of progeny tested per sire and the increased probability of equitable representation of the progeny across macroenvironments.

In designing a selection experiment or selection program, the number of sires, the number of dams mated to each sire, the number of progeny housed per dam, the selection intensity, the expected inbreeding, and the duration of the experiment must all be considered (Hill, 1980). Facilities and budget are major practical factors that often greatly narrow theoretical options. The optimums with regard to the number of sires, the number of dams mated to each sire, and the number of progeny housed per dam are similar to those for genetic parameter estimation; the reader is directed to Robertson (1959a, 1959b) and Hill (1980) for a discussion. However, the reader is cautioned that the formulae are theoretical and approximate for balanced designs, and designs with very small family sizes are likely to deviate more from these expectations than those with larger family sizes.

In practical terms, it is not possible to achieve a perfect balance in terms of the number of dams mated to each sire and the number of progeny housed per dam. Infrequently, a sire is 'lost', but in populations where fertility, hatchability and viability are considered in the selection program, this will be a minor problem. With the swift replacement of selected female breeders that go out of lay in the early part of the egg save period, the loss of dams can be minimized. The biggest imbalance occurs due to differences caused by variation in the number of progeny per dam due to differences in rate of lay during the egg save period, fertility, hatchability, viability, sex ratio, and mortality. The impact can only be minimized by planning that must start at selection.

Population size is important with regard to the effectiveness of selection, the repeatability of selection responses, and genetic drift. Larger populations are needed for practical selection schemes that are expected to achieve long-term results on a par with commercial schemes than are required for some experimental purposes.

Even though selection of males for the primary traits is based upon the performance of their female siblings, the number of males available in each family can modify the effectiveness of selection schemes, especially if different selection schemes are being compared. If only one male is available for selection per full-sib family, the effectiveness of selection on the male side of the pedigree will be diminished.

It is doubtful whether valid useful results can be expected from experiments where the populations are fragmented into small replicates to better estimate drift variance. This fragmentation can result in poor population structure (i.e. too few dams per sire and too few progeny per sire for the half-sib family mean to be much more reliable than the full-sib family mean or individual phenotypes) for comparison of methods or to expect much absolute gain, and the drift variance may be too large with respect to expected selection response.

Concluding Remarks

Laying stocks of chickens provide one of the best models to test theory and breeding criteria. Some of their advantages are intrinsic economic value, a number of antagonistic and positive relationships among economically important traits, short generation interval, and relatively low cost. Breeding research will not provide all of the answers, but careful well-planned breeding research still pays large dividends; without it many other types of research would not be possible or would have limited impact. Research on disease and disease resistance, management, physiology, etc. done with experimental breeding flocks has been invaluable. Also, without breeding research, the approaches and applications of molecular genetics research may be more limited in scope.

With reduced support for research, there has been pressure to get answers quickly and to abbreviate research studies. In poultry breeding research, this is reflected in the length of selection studies and the length of records maintained. When short-term and theoretical studies have narrowed the techniques and criteria to a few that seem reasonable, the only way to determine the best is empirically with a long-term selection study. Quantitative genetics is a branch of biology, and theory must be proven, amended and refined based on actual biological results. Elegant theory provides hypotheses to be tested. It does not provide definitive answers.

The ultimate goal with regard to egg production is to improve the economic efficiency of egg production over the whole laying period, and indeed over the full life cycle. Where hens are molted for one or more cycles of production, this whole record may be longer than a single cycle of egg production. The end of the egg production cycle is just as important as the beginning, and perhaps it has more importance in cases where adverse effects are more likely to be expressed or maximized near the end of the laying cycle. In many studies egg production has been terminated at about 300 days or earlier. In the United States at present a large proportion of the laying hens are molted at about 420 days of age, so there is some justification for scientists and breeders there in terminating egg records at about the time that the average molt cycle begins. However, a global view would show that in much of the world, and even for a significant fraction of American flocks, a longer record is important. Other than research on molting itself, the important research on extended performance has not been done. The little research that has been published usually studied only one cycle after a single molt, but demonstrated that knowledge of second cycle performance was important (e.g. Fairfull, 1982; Gowe and Fairfull, 1982a).

It no longer seems useful to speculate on the possibility of improving egg weight and egg production (rate or numbers) simultaneously. Overwhelming evidence shows that it is not at all difficult to improve egg weight and egg production together. In practice, the addition of other egg quality traits having several antagonistic relationships among them will still allow significant genetic progress. However, the optimal selection scheme, the optimal combination of traits, and the optimal selection record (especially for multiple-cycle production) are not known.

Prediction of genetic gains is relatively poor in single-trait selection for egg production, with genetic gains usually being overestimated. In multiple-trait selection studies involving egg production, overestimation of genetic gains is much less pronounced. However, retrospective analyses show that the weights placed on different traits often deviate from that intended. There are several reasons that this is so:

1. Many early studies utilized relatively poor environments with respect to aspects of disease and management that are now taken for granted (i.e. range brooding and rearing, and no vaccines for diseases that are now controlled).
2. For many studies, additive genetic variation was poorly estimated.
3. There was a poor choice of an estimator of genetic gain.
4. In gross terms, genetic theory is satisfactory, but many assumptions are either only approximations of the true situation or not valid in all cases.

Theoretical studies attempting to predict selection results should use h^2 and r_g estimates from selected lines. Actual selection results more closely resemble those that would be expected based on parameters in selected as opposed to unselected strains. For example, in unselected strains the r_g of egg production with mature body weight is positive and moderately high, but selection results indicate that body weight usually declines with selection for high egg production, as would be predicted by r_g estimated in selected lines.

Poor environments, especially those with a high disease exposure, are not conducive to genetic progress or accurate estimates of genetic gain for egg production. In selection programs where there is little, none, or negative genetic progress, an environment characterized by poor fertility and hatchability, poor growth and, especially, high mortality is often the root cause. Deliberate use of poor environments is generally a losing strategy, especially with respect to disease where response to each pathogen is specific. The scientist and breeder should ensure that management is as good as possible if maximum genetic gains and valid comparisons are to be made. It bears repeating that: "It is futile to select stocks to perform under bad management whatever the general environment. To make reasonable genetic progress, breeders must be good managers within the economic realities of current management knowledge." (Gowe and Fairfull, 1982b).

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Chapter 30. GENETICS OF EGG PRODUCTION IN OTHER GALLIFORMS

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Selection for increased egg production in the domestic fowl may result from two different objectives. The first involves selection for maximum egg numbers in small-bodied stocks for table egg production, whereas the second involves selection of broiler breeders for the maximum production of settable eggs to produce broiler progeny. Selection for egg production in other galliforms, in general, is more closely related to the latter objective. Therefore strategies for increasing egg production in other galliforms have been less complex than selection for egg production in the chicken.

Information regarding the quantitative inheritance of egg production in avian species other than the chicken is limited. However, reports regarding genetic parameters for egg production in turkeys cover a span of 50 years. Available data are in most cases based on limited population size, small numbers of generations of selection, or only on selected populations. Estimates of genetic parameters for egg production in other galliforms (Japanese quail, guinea fowl, and ring-necked pheasants) are extremely sparse. The high environmental component associated with measures of egg production further complicates the obtainment of a valid base of knowledge regarding the quantitative inheritance of egg production in other galliforms.

EGG PRODUCTION IN TURKEYS

Egg production characteristics. Under natural conditions, turkey hens are strictly seasonal breeders which start to lay in March and April, obtain peak production in late April or May, and gradually cease egg production by October. However, turkeys can be photo-induced to lay eggs during any season of the year provided they are old enough to respond to light. Environmental light is a particularly good synchronizer of physiological events and artificial light with precise control of daylength is utilized to provide continuous poult production throughout the year. Normally, light restriction for eight weeks prior to photostimulation of physically mature hens (30-32 weeks of age for large whites and 28-30 weeks of age in small whites) will result in the onset of egg production within 3-4 weeks following photostimulation with 15-16 h daylengths. After the onset of lay, egg production is normally viewed as egg number from onset of lay, percent hen-day, percent hen-housed, or percent survivor egg production.

Heritability of egg production. Several investigators initially studied the inheritance of egg production in turkeys without estimating quantitatively the heritability of this trait. Asmundson (1938), from an investigation of the many factors affecting egg production in turkeys, reported that these factors were likely influenced by a number of genes similar to those in chickens. Early and late maturing lines of turkeys were developed by Asmundson (1941) and these lines were noted to differ not only in early lay, but also in lay throughout the year. Differences in egg production between four of the more common varieties of turkeys were observed by Whitson et al. (1944). Wilson and Johnson (1946) reported a heritability of .02 from offspring-dam regression for egg production based on pooled data from Bronze and Beltsville Small White turkeys. However, estimates for the Beltsville Small White turkeys were considerably higher than those for Bronze turkeys. This estimate was considerably smaller than the estimate of approximately .40 reported for egg production in Beltsville Small White turkeys by Shaklee et al. (1952).

Blow and Glazener (1954) estimated the heritability of egg production over a five-year period in a population of Bronze turkeys. An average estimate of .13 was obtained from phenotypic correlations between half-sibs, phenotypic correlations between full-sibs, and from the intra-sire regression of offspring on dam. Utilizing a strain of White Holland turkeys, McCartney (1955) reported a heritability based on four generations of data from paternal half-sib correlations of .29 for survivor egg production. Populations of Bronze, Broad-breasted Bronze, Beltsville Small White and crosses of Bronze and Broad-breasted Bronze turkeys were used to study the heritability of egg production by Kondra and Shoffner (1955). They reported heritabilities for egg production ranging from -.65 to .13 with a mean estimate of .13 from combined analysis. Blow et al. (1958) reported heritability estimates for egg production in a Bronze turkey population of .06 for part records and .14 for full records. McCartney (1962) working with a randombred control population of white turkeys (developed by combining four strains) reported a heritability of .49 for egg numbers based on full-sib correlations. In this particular study, paternal estimates (4S) were negative, whereas maternal estimates (4D) were greater than one.

Cook et al. (1962) summarized 12 generations of data obtained from a population of Broad-breasted Bronze turkeys and observed heritability estimates for egg production of .19 and .29 from 4S and 4D components of variance, respectively. Clayton (1962) reported heritability estimates for egg production during the first six weeks in two turkey flocks ranging from .10 to .31 when estimated by full-sib analysis of variance over a two-generation period. Pooled heritability estimates across the two generations were .19 for egg production and .22 for poult number.

Selection for egg production. Initial selection studies for egg production in turkeys indicated the presence of considerable genetic variation, which suggested that this trait was readily amendable by selection. Knox and Marsden (1954), although not presenting any heritability estimates, reported that Beltsville Small White turkeys responded rapidly to selection for increased egg production. These authors reported a doubling in egg production over a seven-year period. However, in the reports of Shaklee et al. (1952) and Knox and Marsden (1954) no nonselected controls were used, and as a result environmental and genetic factors were confounded. Therefore the sharp increases in egg production may have been at least partially due to positive environmental trends.

McCartney and co-workers (1968) were perhaps the first researchers to conduct a selection study for increased egg production in turkeys which included a nonselected control to measure environmental variation. Utilizing the Ohio Randombred Control (McCartney, 1964) as a base population, selection was conducted for five generations for increased egg production. Realized heritability (regression of genetic gain on the accumulative selection differential) for 84-day egg production was .61±.12 (McCartney et al., 1968). The heritability of egg production in this line, obtained from the variation within and between full-sib females, was .32±.15 (Nestor et al., 1972). Continued selection in the same line for seven generations resulted in an increase of 18 eggs per hen in 84-day and 37 eggs per hen in 180-day egg production (Nestor, 1971). The realized heritability of 84-day egg production obtained by regressing the genetic gain on the accumulative selection differential was .33±.05 and the regression was curvilinear ($P<.05$) (Nestor, 1971). The decline in response in later generations was thought to be the result of environmental changes occurring in the randombred control and a slight increase in broodiness in the selected line during these generations.

Clayton (1971) reported that the first 12 weeks may be a more satisfactory measurement of total egg production than the first six weeks and suggested a form of selection based on part record at 12 weeks followed by retrospective culling at 18 weeks. He noted that this system combines the advantage of an extended more accurate measurement period and overcomes most of the difficulties of the two-year cycle. He reported that body weight at different ages or changes in weight between these ages has little effect on egg production and suggested that high body weight *per se* is not necessarily incompatible with high egg number.

A mean heritability of .29±.11 was obtained from three years' data in large-bodied strains and five years' data in medium white strains (Nestor, 1972). Estimates were obtained from regression of daughter on dams, adjusting the data for line effects. Nestor (1972) also found various measures of broodiness to have moderate to high heritabilities (.33 to .46). Two sublines were established from a large-bodied randombred control population by selecting for increased clutch length and decreased total days lost from broodiness (Nestor, 1980a). The realized heritability for average clutch length was .27±.10, while that for total days lost from broodiness was .31±.12. Realized genetic correlations between these two variables were -.60±.56 and -.36±.60.

Nestor (1980b) investigated the influence of a change in management on response to selection in a population which had apparently plateaued for egg production. In generations 10 through 18 of selection, no hens that had gone broody during the laying cycle were used as breeders for subsequent generations. After this change in management, there was a large increase in egg production in the selected line based on both mean egg production and on egg production relative to the control line. The 180-day egg production increased 42 eggs per hen during the nine generations of selection, while there was an increase during the same period of 20 eggs per hen for 84-day egg production.

Changes in total egg mass (average egg weight x average egg production) and biological efficiency (ratio of egg mass to metabolic body weight) were measured in generations 11 to 24 in turkeys selected for increased egg production (Nestor and Bacon, 1986). Although there was a decline in egg weight accompanying the 3.98 egg increase per generation, total egg mass increased .27 kg per generation. Although biological efficiency increased, it did so at a lower rate than the increase in egg production.

Nestor et al. (1987) investigated the influence of selection for increased number of settable eggs produced by turkey hens housed in individual cages. Selection was effective in increasing the number of settable eggs, but the gains were small (3.2 to 5.6 eggs per hen per line over a 112-day production period in eight generations) due to low selection intensity and low realized heritabilities (.10 to .21). The authors attributed these small gains to the fact that the expression of broodiness was suppressed when selection was based on cage performance. As time lost from broodiness is highly correlated genetically with total egg production (-.97) and is highly heritable (.31), any environment that reduces the expression of broodiness would probably reduce selection gains in egg production (Nestor, 1980b).

Egg production — summary. Heritability estimates for egg production in turkeys (Table 30.1) are within the range of corresponding estimates for egg production in the chicken (Kinney, 1969). The mean (.22) of estimates in Table 30.1 for egg production in turkeys is almost identical to average heritabilities in chickens for short, intermediate, and long-term survivor egg production (.22, .19, .22, respectively) reported by Kinney (1969).

Table 30.1. Heritabilities for egg production in turkeys estimated by paternal (4S), maternal (4D), half-sib correlations, and by daughter-dam regression.

Variety[1]	Gen.	Sires	Progeny	h^2	Method[2]	Reference
SBB+BSW	3		514	.02	R	Wilson and Johnson (1946)
BSW	7			.40	S+D,R	Shaklee et al. (1952)
SBB+BBB	5	30	1095			Blow and Glazener (1954)
B,BBB,BSW	3			.13[3]	R	Kondra and Shoffner (1955)
WH	4	27	670	.29	4S	McCartney (1955)
SBB	8	50	2169	.06[4]	4S	Blow et al. (1958)
SBB	8	50	2169	.14[5]	4S	Blow et al. (1958)
C	2	84	446	.49	S+D	McCartney (1962)
BBB	12	67	3043	.19	4S	Cook et al. (1962)
BBB	12	67	3043	.29	4D	Cook et al. (1962)
SBB	2	27-30	316	.21	S+D	Clayton (1962)
LW	2	29-35	364	.16	S+D	Clayton (1962)
LW	5			.32	S+D	Nestor, et al. (1972)
LW, MW	3-5			.29	R	Nestor (1972)
Average				.22		

[1] Variety: C = control population, BBB = Broad-breasted Bronze, BSW = Beltsville Small White, SBB = Standardbred Bronze, WH = White Holland, LW = large white, MW = medium white.

[2] Method: R = regression of offspring on dam, 4S = sire component, 4D = dam component, S+D = full-sibs.

[3] Average of four strains.

[4] Part record - 30 d.

[5] Full record - 100 d.

Selection responses for egg production reported by McCartney et al. (1968) and Nestor (1971) clearly demonstrate that genetic progress can be made in improving egg production in turkeys. The high initial realized heritability (.61) reported by McCartney et al. (1968) and a much lower estimate (.33) reported by Nestor (1971) following continued selection in the same line are in agreement with theoretical expectation that the greatest genetic progress occurs during initial generations of selection, followed by a gradual decline until the response becomes negligible (Lawrence, 1964). This pattern of high initial response to selection followed by a subsequent decline was also observed in selection for high body weight in Japanese quail (Marks, 1978).

Egg weight. The heritability of egg weight in turkeys was found to range from .50 to .64 depending on the method of calculation (Blow and Glazener, 1954). Analysis of variance estimates using 4S and 2(D+S) methods were .53 and .50 respectively. The heritability obtained by regression of offspring on dam was .64. These data were obtained over a five-year period and involved Standardbred and Broad-breasted Bronze turkeys. Heritability estimates for egg weight in a strain of White Holland turkeys were reported by McCartney (1956). The heritability estimate from full-sib correlations was .42, whereas analysis of variance estimates from sire and dam component analyses were .22 and .62 respectively. Nestor et al. (1972) also obtained an estimated heritability for egg weight of .42±.14 from the variation within and between full-sib females.

These data (Table 30.2) indicate that the heritability of egg weight in turkeys is moderate to high and falls within the range of heritabilities generally reported for egg weight in chickens (Shoffner and Sloan, 1948; Kinney, 1969). Irregular oviposition and the production of a large percentage of abnormal eggs in meat-type turkeys has been reported by Nestor et al. (1970). This condition is similar to that reported in broiler strains of chickens (Jaap and Muir, 1968) and further supports genetic similarities between chickens and turkeys in egg formation and weight.

Correlation of egg production with other traits. The consequences of selection for improved egg production in turkeys are similar to those observed in chickens. Estimates of genetic correlations between characters derived from the sire components of variance indicate there may be a negative genetic correlation between egg production and fertility (Cook et al., 1962). McCartney (1962) also observed small negative correlations between egg production and fertility in a randombred control population of turkeys. However, Nestor et al. (1972) obtained a positive genetic correlation of .59±.21 between egg production and fertility.

Genetic and phenotypic correlations between egg production and egg weight in a randombred turkey population were .20 and .06 respectively (McCartney, 1962). A genetic correlation of -.37±.23 between egg production and egg weight was observed by Nestor et al. (1972). Strong and Nestor (1980) found that selection for increased egg production resulted in a major decrease in egg weight which was associated with the production of relatively less albumen. The phenotypic correlations between egg production and egg weight were -.15 and -.11 in medium and large turkeys respectively. Corresponding correlations between egg production and albumen weight were -.19 and -.17.

McCartney et al. (1968) reported that egg production of a growth selected line was not greatly different from the egg production of the randombred control. Realized genetic correlations between eight-week body weight and egg production and 24-week body weight and egg production averaged -.04±.22 and -.14±.10 respectively. Nestor (1977), however, reported larger negative relationships between egg production and body weight. Realized genetic correlations between female 16-week body weight and egg production in two lines were -.42 and -.66 respectively. Although most of the reports showing a low degree of

Table 30.2. Heritabilities for egg weight in turkeys estimated by paternal (4S), maternal (4D), and half-sib correlations, and by daughter-dam regression.

Variety[1]	Gen.	Sires.	Progeny	h^2	Method[2]	Reference
SBB,BBB	5	30	1095	.53	4S	Blow and Glazener (1954)
SBB,BBB	5	30	1095	.50	S+D	Blow and Glazener (1954)
SBB,BBB	5	30	1095	.64	R	Blow and Glazener (1954)
WH	3	18	274	.22	4S	McCartney (1956)
WH	3	18	274	.62	4D	McCartney (1956)
WH	3	18	274	.42	S+D	McCartney (1956)
LW	5			.42	S+D	Nestor et al. (1972)
Average				.48		

[1] Variety: BBB = Broad-breasted Bronze, SBB = Standardbred Bronze, WH = White Holland, LW = Large White.

[2] Method: R = Regressions of offspring on dam, 4S = sire component, 4D = dam component, S+D = full-sib.

association between body weight and egg production utilized small birds, Clayton (1971) found a small nonsignificant negative genetic correlation between body weight and egg production in birds that were similar in body weight to the lines utilized by Nestor (1977). Arthur and Abplanalp (1975) reported genetic correlations between body weight and egg production varying from -.43 to .24 in six flocks originating from the same base population.

Nestor (1985) developed a line (FE) tandemly selected for both increased 16-week body weight and increased egg production from a line F that had previously been mass selected for 11 generations for increased 16-week body weight. Following four generations of selection for egg production and two generations for body weight, results obtained indicated that the genetic correlation between 16-week body weight and egg production was negative. Body weight declined during the phase of selection for increased egg production, while egg production declined during the last two generations of selection for increased body weight. The author suggested that a part of the apparent negative genetic correlation could possibly be explained by a decline in the mean due to the effects of relaxed selection. Previous studies have shown that the genetic correlation between body weight and egg production varied depending on the point in selection when the correlation is measured (Nestor, 1971, 1977, 1981, 1984). Nestor (1985) concluded that because hens of the FE line laid only a few more eggs, selection for increased body weight alone would be more economical in an integrated operation than selection for both traits by use of tandem selection.

EGG PRODUCTION IN JAPANESE QUAIL

Marks and Kinney (1964) reported heritability estimates of .38 and .40 for egg number in two populations of Japanese quail. Pooled heritabilities across populations were .36 and .46 for age at first egg and percent hen-day egg production respectively. Sittman et al. (1966) estimated the heritability of egg production from first egg to 12 weeks of age, from 12-16 weeks of age, and from first egg to 16 weeks of age to be .20, .09, and .36 respectively. The heritabilities of laying rate (10-13 weeks of age) estimated by analysis of variance and by intra-sire regression of offspring on dams were .24 and .10 respectively in a randombred population (Sato et al., 1980).

Strong et al. (1978) conducted a study to examine the inheritance of egg production, egg weight, body weight, and certain plasma constituents in a randombred quail line. Egg weight and 70-day egg production were estimated to have moderate to high heritability (sire component estimates ranged from .26 to .50) and were influenced greatly by maternal and/or nonadditive genetic effects (Table 30.3). Phenotypic correlations between egg production and egg weight were from .03 to -.11, between egg weight and body weight from .26 to .46, and between egg production and body weight from .04 to .06. The genetic correlations between egg weight and egg production were negative (-.35 and -.07) and larger between early egg weight and production than for egg weight at an older age (Table 30.4). Genetic correlations between egg production and body weight were also negative (-.05 and -.04), whereas correlation of body weight and egg weight were positive and of moderate to high magnitude (.42 to .54). Correlations between growth and reproductive traits were investigated in Japanese quail by El-Ibiary et al. (1966). Significant phenotypic correlations were found between egg number and adult body weight (.34) and between egg number and sexual maturity (-.64). Egg number and egg size seemed to be unrelated. The relationships between egg production and fertility and hatchability, and between egg weight and fertility and hatchability, were small and not consistent. In chickens and turkeys egg

Table 30.3. Heritability estimates ± standard errors of egg weight, egg production, and body weight of Japanese quail. (Modified from Strong et al., 1978).

	Heritability estimate		
Trait	Sire component	Dam component	Sire + dam
Egg wt. 1	.50 ± .42	1.07 ± .25	.78 ± .22
Egg wt. 2	.28 ± .39	.71 ± .26	.50 ± .20
70 day egg prod.	.26 ± .37	.58 ± .26	.42 ± .20
Body wt. 1	.02 ± .45	1.16 ± .28	.59 ± .23
Body wt. 2	.10 ± .45	1.16 ± .28	.63 ± .23

Table 30.4. Genetic correlation estimates ± standard errors obtained from full-sib (sire + dam) components of covariance. (Modified from Strong et al., 1978).

	Egg wt. 2	70-day egg prod.	Body wt. 1	Body wt. 2
Egg wt. 1	.80 ± .57	-.35 ± .44	.47 ± .31	.43 ± .33
Egg wt. 2		-.07 ± .54	.54 ± .31	.42 ± .36
70-day egg prod.			-.05 ± .13	-.04 ± .14
Body wt. 1				.98 ± .00

weight tends to be negatively correlated with egg production and positively correlated with body weight (Kinney, 1969; Nestor et al., 1982).

Nestor et al. (1983) reported selection responses in quail lines divergently selected for high (HE) and low (LE) 120-day egg production based on individual egg records. After five generations of selection, realized heritabilities for HE and LE were .06±.06 and .35±.02 respectively. Selection decreased egg production in the LE line by 19 eggs per hen, whereas a gain of only three eggs per hen was realized in the HE line. The authors noted that this asymmetrical response was not totally unexpected because the base population had high egg production, ranging from 109 to 113 eggs in 120 days. In addition, the selection differentials were more than twice as large in the LE as in the HE lines (10.5 vs. 4.5 eggs). Egg weight was not significantly changed in the HE and LE lines during the five generations of selection for high and low egg production. The results of this selection study suggest that although considerable genetic variation is present in quail for egg production, most of the variation is associated with low egg production.

EGG PRODUCTION IN GUINEA FOWL

Information on quantitative egg production parameters in guinea fowl is unavailable. Although guinea fowl have not been selected as intensively for egg production as have chickens, turkeys, and Japanese quail, it appears that they have considerable genetic variation for this trait. Wild guinea fowl have clutch sizes of 6-10 eggs (Long, 1981).

Under proper temperature and light environments, Hughes (1986) reported hen-day egg production above 70 percent after 16 weeks of lay. Oguntona and Zubair (1987) reported a mean number of 120 eggs per hen per year over a four-year period, with egg numbers greater than 150 eggs per year in the first two years. In France, under commercial conditions, guinea fowl normally lay 160 eggs per bird during a laying period of 34-36 weeks. Therefore, it appears that egg production in the guinea fowl has been improved dramatically and that genetic parameters for egg production traits are likely similar to those observed in other domestic avian species.

EGG PRODUCTION IN RING-NECKED PHEASANTS

Gooding (1955) suggested that pheasants will lay 50 or more eggs in a season with average production of about 35 eggs. Sheppard and Flegal (1973) reported average hen-housed egg production per bird for three strains varied from 33 to 61 eggs. They suggested that these results indicated a wide variation in the egg production potential of the ring-necked pheasant.

In the early 1970s a project was initiated at Michigan State University to try to increase production of eggs from ring-necked pheasant breeder hens. At the beginning of the project in 1970 and 1971, egg production was quite low, with 32.9 percent hen-housed egg production during a 120-day production period (Wing, 1976). However by 1976, egg production had risen to 52.7 percent per hen housed for the 120-day test period. Wing (1976) attributed most of the increase in egg production to improvements in management procedures and the realized heritability was estimated to be only .07. In 1977, the housing system changed from one of floor breeding flocks to that of caging the females. This increased the number of breeders and allowed a greater degree of selection pressure (Carpenter, 1980). Percent hen-housed egg production (120-day) increased to approximately 66 and 68 percent in 1978 and 1979 respectively.

Fathy (1982) reported that selection for increased egg production in ring-necked pheasants resulted in an average improvement of two eggs per year and they reported a realized heritability for egg production of .24. Heritability for egg production from sire, dam, and sire + dam variance component analyses yielded estimates of .03±.15, .41±.22 and .26±.07 respectively. Regression of daughter on dams (egg production) resulted in a heritability of .38±.38. Fathy (1982) found the phenotypic and genetic correlations between egg production and other traits to be small in ring-necked pheasants.

CONCLUDING REMARKS

Quantitative genetics of egg production parameters in other galliforms (turkeys, Japanese quail, guinea fowl and ring-necked pheasants) generally appears similar to corresponding parameters in the domestic fowl (Table 30.5). However, since these species have not undergone intense selection pressure for egg production traits, there appears to be considerable additive genetic variation remaining for these traits. Therefore, individual phenotypic selection should result in substantial initial increases in egg production. Long-term selection strategies will, however, likely require family selection or progeny testing systems similar to those used for improvement of egg production in chickens.

A relatively high realized heritability has been observed for egg production in turkeys which may be the result of selection pressure for both increased egg number as well as

Table 30.5. Summary of mean heritabilities for egg production in other galliforms.

Species	Method[1]	h^2	Range	Estimates, no.
Turkey	E	.22	.02 to .49	13
	R	.47	.33 to .61	2
Japanese quail	E	.32	.09 to .58	11
	R	.21	.60 to .35	2
Ring-necked pheasant	R	.16	.07 to .24	2

[1] Method: E=estimated from paternal and maternal half-sib correlations and parent regression, R=realized.

decreased incidence of broodiness.

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Chapter 31. GENETICS OF EGG PRODUCTION AND REPRODUCTION IN WATERFOWL

H. Pingel

Reproduction is a subject of continuing interest in waterfowl breeding as a means of increasing the number of ducklings and goslings for meat production. There are negative correlations between early growth rate and subsequent egg production and hatchability, and therefore growth and fecundity cannot be incorporated into a single population. As a consequence reproductive performance has to be improved in specialized female strains. In some parts of the world duck egg consumption also plays an important role; for example, in southeast Asia duck eggs account for about 25 percent of total egg consumption. Besides native breeds, Khaki Campbell ducks and their crosses are used for the production of eggs for human consumption.

EGG PRODUCTION TRAITS

The reproductive performance characteristic of domestic waterfowl species is shown in Table 31.1. There are great differences among species in the important traits.

Laying intensity and persistency are the main factors affecting egg production. The laying intensity depends on the interval between consecutive ovipositions. In the study of Simmons and Hetzel (1983) using Khaki Campbell ducks, it was found that oviposition generally occurred between 4 a.m. and 7 a.m., 7-10 h after the onset of the dark period. The mean time interval between consecutive ovipositions was 24.0 h with a range of 23.5-24.5 h. It was estimated that ovulation occurred on average ten min after oviposition. The ovum spent 15-30 min in the infundibulum, 2.5-3 h in the magnum, 2-2.5 h in the isthmus and 18.6 h in the shell gland. Stasko and Masar (1969) investigated some characteristics of egg laying in geese. The geese of different breeds lay most of their eggs between 9 p.m. and 5 a.m. (60.4 percent); 38.1 percent were laid between 5 a.m. and 3 p.m., and 1.5 percent between 3 and 7 p.m.

Short persistency of lay adversely affects egg production in geese and muscovy ducks.

Table 31.1. Characteristics of reproductive performance in waterfowl.

	Pekin ducks	Muscovy ducks	Geese
Age of first egg, d	180	210	260
Duration of laying, d	280	150	130
Laying rate, %	75	50	40
Eggs produced, no.	210	75	50
Fertility, %	90	80	80
Hatchability, %	80	75	75
Offspring, no.	150	45	30

Under natural light in the spring, egg production is increased resulting from the stimulus provided by daily increments in the hours of daylight. A few months later, egg production will decrease and then will cease. This is due to a regression of gonads under long daylengths. Limitation of the lightday to ten hours prolongs laying persistency and markedly increases the egg number of geese (Schneider, 1981).

Broodiness has been eliminated in the most important breeds of waterfowl, but in some local breeds it is a normal part of the process of reproduction and it lowers egg production.

Heritability estimates for various egg production traits are shown in Table 31.2. The data show that the heritability for egg number is low. An exception is the heritability of egg number from two-year-old geese estimated by Wezyk and Sochocka (1978) and the part-period egg production from ducks estimated by Pingel (1976). The higher h^2 value for egg number in the first part of the laying period (from first egg to March 31) could be due to a lower environmental component. The heritability coefficients for egg weight were rather high with a range from 0.35-0.77. The heritability for age at first egg was medium (0.34-0.49). The genetic variability for this trait can be reduced by short daylength and by a restricted feeding system, and then the genetic potency will be masked.

In general there is a negative correlation between early growth rate and subsequent egg production. In rapidly growing strains of ducks and geese it has been observed that the ovary produces more than one yolk each day resulting in soft-shelled or membranous eggs or double-yolked eggs which cannot be used for hatching. Table 31.3 shows low positive or negative correlations between body weight and egg yield. Pingel (1976) has estimated genetic correlations in two Pekin duck strains of r_g=-0.26 and r_g=0.002.

Effects of selection. Selection for egg production has profoundly changed the physiology of egg laying in ducks. Hutt (1952) reported that some Khaki Campbell ducks were found to produce eggs without interruption for 418 consecutive days. At the Cherry Valley (UK) breeding farm, hybrids of duck strains specialized for the production of table eggs lay 275 eggs from 20 to 72 weeks of age, and meat-type duck parent stocks can produce more than 200 eggs in a 40-week period (Powell, 1985). Grimaud, the leading breeder of muscovy ducks in France, increased egg production from 110 to 170 eggs per bird during two periods of five months (Retailleau, 1985). Merritt (1962) increased the egg number of Pilgrim geese from 20 in 1951 to 36.8 in 1960. From 1961 to 1970 Stasko (1981) doubled the egg yield per goose. But in both cases the laying intensity was at a constant level of about 40 percent. The increase in egg number was due to prolonging of the laying period.

There is a belief that a decrease in heritability level should take place as a consequence of selection within a closed flock. Wezyk and Sochocka (1976) selected for increased egg number in two strains of Italian geese over six generations. In strain W 11 egg number was increased from 24.8 to 38.0 eggs and in strain WD 1 from 44.3 to 48.3 eggs. In subsequent generations the heritability of egg number in both strains was low, but it did not decline as expected; heritability for egg production in strain W11 amounted to 0.15, 0.59, 0.40 and 0.58 and in the strain WD 1 to 0.56, 0.24, 0.18 and 0.39 respectively. The heritability of egg weight also did not show a distinct trend over time.

To increase selection efficiency, Stasko et al. (1976, 1979) have considered using part-period records. But it must first be resolved whether the egg number of the first laying period is a reliable index of egg number in the following season. Generally egg production of geese is higher in the second and third year, and it begins to fall at the age of four to five years. Merritt and Lemay (1963) and Stasko and Masar (1969) have found the highest rate of laying in the second laying year. The correlation coefficient between first and second

Table 31.2. Heritability of egg number (EN), egg weight (EW), and age at first egg (AE).

Breed	Trait	Number of			Av	h^2_{S+D}	Reference
		Sires	Dams	Offspring			
Pekin ducks	EN	69	168	1169	132	0.32	Stasko (1968)
	EW	69	168	1169	83	0.47	
Pekin ducks	EN	36	186	1129	125	0.26	Sochocka and Wezyk (1971)
	EW	36	186	1129	86	0.47	
	AE	36	186	1129	282	0.34	
	EN to March 31			2393	49	0.42	Pingel (1976)
	EW to July 31			2393	142	0.23	
	EN Apr 1-July 31			2393	93	0.17	
	EN	44	215		167	0.23	Cerveny et al. (1986)
	EW	44	215		88	0.40	
	EN				153	0.17	Mazanowski (1988, personal communication)
	EW				88	0.60	
Muscovy ducks	EN	140	321	2897	65	0.16	Ricard et al. (1983)
	AE	140	312	2897	231	0.49	
Italian geese	EN	26	87	318	37	0.24	Stasko and Masar (1969)
	EW	26	87	318	153	0.40	
	EN	225	633	1520	41	0.37	Wezyk et al. (1975)
	EW	225	633	1520	157	0.67	
	EN 1st yr	56	152	412	43	0.18	Wezyk and Sochocka (1978)
	EW 1st yr	56	152	412	155	0.71	
	EN 2nd yr	54	133	276	56	0.61	
	EW 2nd yr	54	133	276	177	0.77	
	EN 3rd yr	54	133	306	47	-0.08	
	EW 3rd yr	54	133	306	182	0.63	

year production calculated by Stasko et al. (1976) was r=0.19. Wezyk and Sochocka (1978) calculated correlation coefficients between the egg number of three consecutive laying periods in geese (Table 31.4). The coefficients were also low, resulting from the observation that geese laying more eggs in the first year tend to lay fewer eggs in the second and third years.

It is not certain whether selection for part-period laying is successful. Correlations between egg number in the first part and the total number of eggs laid in the whole period are high and positive. That is because the eggs laid in a short period are a more or less large part of the total number laid in the whole period (Table 31.5). Therefore it can be concluded that egg number in the first part is of less value in predicting egg number of the residual part of the laying period. Direct selection for the part-period egg number, if successful, would result in an increase in egg number for this period. If there is a low relationship between egg production in the first part and in the residual part of laying period, the increase in total production would be equivalent to the increase in the part-

Table 31.3. Phenotypic (r_p) and genetic (r_g) correlations between body weight and reproductive traits.

		Body weight at the age of			
		8 weeks Pekin ducks	6 weeks Pekin ducks		8 weeks Italian geese
			line 13	line 24	
Sexual maturity	r_p	-0.13			
	r_g	-0.17			
Egg number	r_p	0.04	0.09	-0.17	0.06
	r_g	-0.08	0.19	-0.42	0.04
Egg weight	r_p	0.32	0.44	0.28	0.38
	r_g	0.40	0.87	0.44	0.47
Hatchability	r_p		-0.01	-0.09	0.02
	r_g		0.02	-0.52	0
Reference		Sochocka and Wezyk (1971)	Hudsky et al. (1986)		Wezyk et al. (1975)

Table 31.4. Correlation coefficients between consecutive laying periods in geese. (Adapted from Wezyk and Sochocka, 1978).

	1st to 2nd	1st to 3rd	2nd to 3rd
Egg number	0.33	0.25	0.39
Egg weight	0.83	0.73	0.80
Fertility	0.09	0.10	0.17
Hatchability	0.21	0.29	0.35

Table 31.5. Correlations between egg number of part-period, residual, and full period in Pekin ducks. (Adapted from Pingel, 1976).

Strain	Part-period to full period		Part-period to residual period	
	r_p	r_g	r_p	r_g
A	0.59	0.70	0.30	0.22
B	0.61	0.63	0.12	0.01
C	0.69	0.98	0.11	0.25

period.

Schneider (1987) has investigated the characteristics of laying in geese. The phenotypic correlations between total number of eggs laid (62.3) and age at the beginning of the laying period (288 days), laying persistency (140 days), and laying intensity (44.6 percent) were 0.23, 0.64 and 0.69 respectively. Therefore it seems necessary to look for selection criteria that would make it possible to increase laying intensity and length of laying period. Laying intensity and age at the last egg in the laying period might be very efficient selection criteria and should be investigated in the future.

REPRODUCTION TRAITS

Reproduction depends not only upon the number of hatching eggs laid but also upon their fertility and upon the amount of embryonic mortality during incubation or before it.

Mating ratio. The traditional close mating ratio is a disadvantage in the reproduction of waterfowl. In experiments concerned with the possibility of reducing the number of male geese in a flock, Bielinski (1979) compared the mating ratios of three to nine females per male. It was found that a wide mating ratio did not negatively influence reproduction results. Ganders of male lines should be selected for the ability to fertilize the eggs of up to nine geese.

Semen traits. Decreased fertilization capacity of ganders is sometimes observed in the second half of the breeding season. Bielinski (1979) called it sexual exhaustion. This phenomenon is caused by poor quality of semen and decreased sexual activity of ganders, accompanied by an increase in body weight and molting. Histological examination of testes of ganders during the breeding season showed that testis weight, the diameter of seminal tubules, and activity of spermatogenic and spermiogenic processes were the highest from February to April. Beginning in May changes in spermatogenesis and degenerative processes in the testes due to long daylength were observed. Ganders exposed to light for 10-12 h had a steadier and larger semen production than ganders under longer daylengths.

Pimenov et al. (1979) found large differences in the amount and in the concentration of ejaculates among ganders collected by the massage method. About 30 percent of ganders did not show an ejaculation reflex.

Stasko and Majna (1970) estimated significant correlations between ejaculate volume collected by the massage method and length of penis (0.54 to 0.24) and semen collection time (-0.49 to -0.10). Heritabilities of penis length (43.8-51.2 mm), semen volume (0.40-0.60 ml) and semen collection time (49.2-53.7 sec.) were 0.32, 0.24 and 0.09 respectively.

The morphology of muscovy spermatozoa was described by Maeda et al. (1984). The average lengths of the segments were: acrosome 1.8 μm, nucleus 10.9 μm, midpiece 3.6 μm, and flagellum 71 μm. Low fertility may be related to an increase in the proportion of spermatozoa with crooked necks and ascrosomal damage.

Duration of fertility. The maximum duration of fertility following artificial insemination was found to be 16 days in Pilgrim geese (Johnson, 1954), 14 days in Emden geese (Kinney and Burger, 1960) and 15 days in Alabio ducks (Setioko and Hetzel, 1984). The relatively short duration of fertility requires a high frequency of insemination.

Fertility and hatchability. Selection for growth rate has a negative effect on hatchability. This was confirmed by the comparison of a heavy and a light strain of Pekin ducks (Vagt et al., 1989). Body weights of the two strains at eight weeks of age were 3.8 kg and less than 2.2 kg. Hatchability was 63.5 percent for the heavy strain and 90.9 percent for the light one. At the beginning of the laying season the frequency of chromosomal aberrations in embryonic tissue was much higher in the heavy strain (14.3 percent) than in the light strain (8.1 percent). This difference was due to more frequent appearance of euploid mosaics. The heavy strain was characterized by slower embryonic development and by a high proportion of dead embryos during the first 40 h of incubation.

Cerveny et al. (1986) estimated the heritability of fertility, hatchability, and number of ducklings per dam in six strains. The coefficients were mostly low and medium values. Heritabilities for fertility were 0.20 to 0.74, for hatchability 0.11 to 0.56, and for ducklings per dam 0.13 to 0.48. Other estimates of heritability for fertility and hatchability are shown in Table 31.6. In most cases low heritability coefficients were obtained for fertility and hatchability, which is in close agreement with other reports. Selection to improve hatchability should consider the length of hatching time.

The estimates of genetic and phenotypic correlations between egg production and reproduction traits are given in Table 31.7. The correlation between egg number and fertility tends in ducks to be positive and in geese to be negative. The correlations between egg weight and both fertility and hatchability are very low.

Selection systems. There are two systems of selection for high reproductive ability in female lines bred as fully pedigreed populations. The typical breeding program is based on 20 breeding pens with one male and 4-5 females. The daughters are trapnested during

Table 31.6. Heritability of fertility (F) and hatchability (H).

Breed	Trait	Number of			Av	h^2_{S+D}	Reference
		Sires	Dams	Offspring			
Pekin ducks	F	45	130	738	88.7	0.17	Stasko (1968)
	H				86.2	0.14	
Pekin ducks	F	44	215		76.9	0.52	Cerveny et al. (1986)
	H				83.2	0.42	
Pekin ducks	F				93.0	0.01	Mazanowski (1988, personal communication)
	H				81.3	0.15	
Italian geese	F	108	299	591	68.9	0.41	Wezyk et al. (1975)
	H				76.7	0.14	
Italian geese	F	55	139	319	73.5	0.13	Wezyk and Sochocka (1978)
	H				82.1	0.29	
Italian geese	F	8	38	144	72.6	0.16	Bielinska et al. (1981)
	H				78.9	0.09	
Czech White geese	F				86.3	0.10	Hudsky et al. (1971)
	H				67.1	0.04	

Table 31.7. Relationships of egg number (EN) and egg weight (EW) with fertility (F) and hatchability (H).

		F		H		Reference
		r_p	r_g	r_p	r_g	
Pekin ducks	EN	0.72		0.66		Hattenhauer et al. (1981)
Pekin ducks	EN	0.89	0.70	0.41	0.29	Hudsky et al. (1986)
	EW	0.05	0.05	0.06	0.13	
Italian geese	EN	-0.18	-0.53	0.10	0.03	Wezyk et al. (1975)
	EW	0.04	-0.13	-0.13	0.08	
Italian geese	EN	-0.38		0.09		Stasko et al. (1976)
	EW	-0.19		-0.29		

the first laying season. Based on these records the best birds are selected for the breeding pens in the second year of life. Alternatively, Pingel (1986) has proposed using retrospective selection. All birds are kept in breeding pens during the first laying season. Based on trapnesting records, information on sexual maturity, laying intensity, duration of laying period, fertility and hatchability is evaluated for the selection of sons and daughters of the best dams. The final retrospective selection is based on full records of reproduction.

Crossbreeding and heterosis. Most commercial duck and goose stocks are multiplied via grandparent and parent stock which produce the hatching eggs for duckling and gosling production. At the grandparent level, two lines are crossed to produce hybrid vigor for reproductive performance in the parent stock. These hybrid female parents are mated to a typical male line or they are backcrossed. The latter method has the advantage that, out of two lines, the line with higher meat yield and lower feed conversion has about 75 percent influence on the final product.

The crossing of two dam lines to utilize heterosis for reproductive traits is of major importance in waterfowl. But there is little information on the effect of crossing on egg production and fecundity. Stasko (1981) found that egg production of reciprocal crosses was higher than for their Pekin parent and almost the same as for their Khaki Campbell parent. Mostageer et al. (1969) also compared Pekin and Khaki Campbell ducks and their reciprocal crosses. The crossbreds reached sexual maturity midway between the pure breeds. The highest egg number was attained by the Khaki Campbell parents but the Campbell x Pekin crossbred had the highest laying intensity and egg mass production. Mazanowski et al. (1978) compared certain reproductive traits of five duck strains and some of their crossbreds. As expected it was found that two-way crosses and especially three-way crosses (having crossbred dams) are characterized by a higher egg number and hatchability resulting in a higher number of ducklings. Table 31.8 shows a part of these results. The highest duckling production per dam was observed in the mating of drakes from line 4 with hybrid 52 dams which contributed the highest maternal heterosis. Schneider (1976) found a high combining ability when crossing ganders of the German laying goose with Italian geese; the number of eggs exceeded the reciprocal cross and the pure breeds by over 30 percent.

The parent stock hatchery is mainly interested in reproductive traits to maximize

Table 31.8. Reproductive traits of strains and strain crosses of White Pekin ducks. (Adapted from Mazanowski et al., 1978).

Strains and crosses	Egg number per duck	Fertility, %	Hatchability, %	Number of ducklings per dam
2	136.7	91.9	72.4	85.8
5	133.6	91.4	78.0	94.3
2 x 5	133.5	95.5	79.6	100.7
5 x 2	127.6	94.8	74.8	89.3
4	137.7	96.4	75.6	99.3
4 x 25	132.5	96.8	80.7	102.1
4 x 52	139.6	94.2	82.3	107.0

income from gosling and duckling sales. Fatteners are interested especially in growth rate and feed conversion. Micek et al. (1986) recommended use of a 'profit function' in classifying and selecting the base populations and their combinations. They have used this profit function to optimize commercial crossing in geese. The profit function reaches its highest values for three-way crosses. The heaviest line with low egg production and maintaining high fertilizing ability proved to be the best as a male line. The best female lines had average reproduction and good body weight. Selection of female lines for very high egg production with a simultaneous decrease in body weight is not effective.

Backcrossing should be taken into consideration in further investigations on use of this profit function. In the first step the specialized sire and dam lines are crossed. Then female hybrids are backcrossed to the sire line. This yields an 'economic heterosis' by providing a combination of low gosling or duckling cost with superior growth and meat proportion.

Mulard production. Crossing muscovy drakes with domestic ducks of Pekin or other breeds often results in poor fertility and hatchability of eggs. This may be attributed to many factors such as physiological incompatibility of gametes, differences in karyotypes, or chromosome rearrangements. In this context the use of artificial insemination (AI) is important. Semen collection from the muscovy drake by massage is difficult, but collection by means of an artificial vagina is very simple and successful for practical use. The semen must be placed immediately after collection into the vaginal orifice. Skill is needed in everting the genital tract to expose the uterovaginal junction, which can be identified by its spiral appearance. Gvaryahu et al. (1984) obtained an average fertility of 48 percent by using AI twice per week. In small flocks however, when the mating ratio was 1:2.5, natural matings have yielded a fertility of 80 percent.

It seems to be necessary to select muscovy drakes with the highest biological predisposition for reproduction by hybridizing. Rouvier et al. (1987) crossed one group of Rouen ducks and two groups of Pekin ducks with muscovy drakes using AI. The fertility percentages of eggs set were 69, 52, and 61 respectively, and the repeatability of egg fertility was 0.70, 0.89 and 0.88. Sawitzki (1987) crossed muscovy drakes with Pekin ducks of different strains to test their combining ability (Table 31.9). Crossing of muscovy drakes with ducks of the Medeo 1 strain gave the best results and was proposed for practical use. These results indicate the possibility of making use of specific combining ability for increasing fertilization and hatchability. In further investigations the combining

Table 31.9. Results of crossing muscovy drakes with Pekin ducks of different strains. (Adapted from Sawitzki, 1987).

	Pekin strains		
	Medeo 1	Medeo 2	K 13
Fertility, %	84.6	58.4	60.2
Hatchability, %	55.4	53.2	51.8
Hatch of eggs set, %	40.0	32.4	29.6
Body weight of mulards, 77 d, g	3496	3271	3110

ability of individual muscovy drakes should be tested to evaluate selection for high fertility and hatchability in the production of mulards.

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Chapter 32. GENETIC VARIATION IN EGG COMPOSITION

K.W. Washburn

Although dietary variation is more important than genetic variation in its effect on the variation in egg composition there is evidence for some genetic influence on composition of proportions of yolk and albumen, albumen quality, cholesterol, fatty acids, vitamins, minerals, deposition of metabolic products and inclusions in the egg, protein and energy content, yolk disorders, and shell characteristics.

The weight of the egg and its components are influenced by a number of genetic and nongenetic factors. These factors may have a direct effect on egg weight or may affect it indirectly by affecting body weight and egg weight. Genetic changes which would result in changes in egg size should be expected to result in changes in the weight of the egg components. If the component parts are not changed proportionally as the egg weight is changed, then a quantitative change in the composition of the egg will result.

The egg white contains a large amount of water and protein, with small amounts of carbohydrates, ash, and a trace of lipids. Since albumen quality would be expected to result from variation in the quantity, quality, or arrangement of the protein portion, variation in albumen quality would be an indirect indication of variation in egg composition.

The relatively high level of cholesterol in the avian egg has become of increasing interest because of concern about the effect of dietary cholesterol on health. Dietary manipulations can decrease yolk cholesterol level but results are not consistent. There is evidence for genetic variation in egg cholesterol suggesting that a genetic approach to a permanent reduction in egg cholesterol is feasible. There is also some evidence for genetic differences in fatty acid content of the egg.

The vitamin and mineral content is rather consistent across genetic groups. There have been studies showing genetic variation in the levels of riboflavin, thiamine, and manganese. The composition of the egg is the end result of numerous metabolic pathways. This metabolism can be genetically influenced resulting in the deposition of metabolic products which give an off-flavor to the egg. Although not a metabolic end product, meat and blood spot inclusions can be deposited in the egg, resulting in an undesirable product. Genetic variation has been shown for the incidence of both blood and meat spots. A small number of studies have shown evidence for genetic variation in the protein content, energy content, and incidence of yolk disorders.

Shell characteristics for which genetic variation has been shown primarily include shell strength and color with some evidence for genetic variation in shell porosity, membranes, and texture.

PROPORTIONS OF YOLK AND ALBUMEN

Avian species can be divided into two classes based on the proportional parts of the egg content (Romanoff and Romanoff, 1949). In precocial species the yolk makes up 30-40 percent of the total egg weight while in altricial species the yolk makes up only 15-20 percent. Genetic variation in proportion of yolk and albumen has been expressed in studies primarily as a percentage, although some studies have measured percentage solids or only weight. Comparisons between genetic groups have included those between pure breeds, commercial layers, layer lines, crosses and families, lines selected for various characteristics such as shell color, body weight and fatness, and between various species. The age at which the determinations were made varied from 20-95 weeks. Egg weight was also variable in the studies.

Genetic variation and association with egg weight. Differences for percentage yolk and percentage albumen and solids content have been shown for nonselected randombred populations, commercial layer strains, inbred lines and selected lines. The percentage yolk was shown by Washburn and Marks (1985) to be lower in a randombred Leghorn population than in the Athens-Canadian randombred population. Significant differences in the egg solids content of different commercial flocks (Rose et al., 1966) and in percentage yolk and albumen of commercial layer strains (Arafa et al., 1982) have been shown. A large range and significant differences for percentage yolk and albumen were found by Abplanalp et al. (1984) in inbred Leghorn lines. The variation was even greater in the two-way crosses which showed evidence of positive heterosis effects for percentage yolk and negative heterosis effects for percentage albumen. Genetic differences in ratio of yolk to egg weight between inbred lines were shown by Erasmus (1954). Significant differences in proportions of egg content of three Leghorn lines which had been selected for rate of egg production, and large or small egg size were shown by Marion et al. (1964). Eggs from White Leghorns selected for economic traits were shown by Rodda et al. (1977) to contain more percentage albumen and less percentage yolk than nonselected controls. The percentage yolk was greater and the percentage albumen lower in eggs from dwarf White Leghorn lines selected for low body weight compared to eggs from a line selected for high body weight (Benoff and Renden, 1983). The White Leghorn breed was shown by Sáinz et al. (1983) to have lower percentage yolk and dwarf Sussex had higher percentage yolk than Rhode Island Reds, Sussex, Maran, or English Gamecock breeds.

In all of these studies in which egg weight was reported, the genetic groups that differed in proportions of egg components also differed in egg size, the group with the larger egg size having relatively less yolk than albumen as compared to groups laying smaller eggs. When the effect of egg size differences between genetic groups was removed the genetic effect was negligible, leading to the conclusion that variation in percentage yolk and albumen is covariable with egg weight. The genetic correlation of yolk weight with egg weight is high (.54 to .62) and that of percentage albumen even higher (.86 to .91) according to Rodda et al. (1977).

There are exceptions to the association of smaller percentage yolk in larger eggs. In one comparison, Cherry et al. (1978) found no significant differences in percentage yolk or percentage albumen between dwarf or normal high and low weight lines whose mean egg weight ranged from 30.7 to 51.1 g. In another comparison, the percentage yolk of the high weight line with larger egg size was greater than that of the low weight line. Scheinberg et al. (1953) found no clear-cut differences between New Hampshire, Barred Rock and White Leghorn breeds in percentage yolk or albumen, although the egg weight ranged from 51.8 to 56.1 g. In the study of Sáinz et al. (1983) the percentage yolk of the English Gamecock breed was lower than that of three other breeds which had much higher egg weight. Although Marion et al. (1965) observed negative correlations between percentage yolk and egg weight within lines, the mean percentage yolk of the five lines used was not negatively associated with egg weight.

Several studies have shown that albumen weight in avian species is highly correlated with egg weight and that most of the variation in egg weight can be accounted for by variation in albumen weight (Jull, 1924; Knox and Godfrey, 1934; Olsson, 1936; Asmundson, 1933; Cherry et al., 1978; Benoff and Renden, 1983; Strong and Nestor, 1980). When eggs were divided within strain into large and small weight classifications in the study of Marion et al. (1964), the larger eggs had less percentage yolk and more percentage albumen than smaller eggs. Strong and Nestor (1980) and Nestor et al. (1982) concluded that the egg weight increases associated with body weight increases in turkeys were due to increases in the weight of the albumen with no change in yolk weights, resulting in a decreased percentage yolk in the large strains.

If genetic variation in albumen amount is greater than that for yolk as suggested by Scheinberg et al. (1953), and if albumen is the main contributor to egg size, then selection for larger egg size would be expected to increase the amount of albumen on a percentage basis to a greater extent than yolk.

Solids content. Because of the consistent association of relatively smaller yolk size with egg size, an association of solids content with egg size might be expected. However, several studies do not support this conclusion. In the study of Marion et al. (1964), percentage yolk solids did not differ significantly between the lines which differed in egg weight and proportions of components. Although the difference in albumen solids was significant, the magnitude of the difference was very small compared to percentage albumen. Varadarajulu and Cunningham (1972) reported that eggs of the Rhode Island Red breed had higher percentage solids than those from a White Leghorn breed in one study, but no differences were found in eggs from two inbred lines of Brown and White Leghorns. Although most of the differences in proportions of yolk and albumen observed by Rose et al. (1966) could be explained by egg weight, they found that some flocks produced large eggs with relatively high solids content. Rodda et al. (1977) found only moderate (.15 to .33) correlations of yolk percentage solids and small (.11 to .17) correlations of albumen percentage solids with egg weight. These studies suggest the possibility that some of the differences in proportions of yolk and albumen observed between genetic groups associated with egg weight may be due to differences in moisture content.

Association with other characteristics. The proportions of the egg components may change in response to selection for other characteristics, particularly those that affect body size, egg size or body composition. The yolk:albumen ratio (Mérat, 1971) or the percentage yolk and albumen (Cherry et al., 1978) were not significantly affected by the *dw* dwarf gene in either a heavy or light weight genome even though the egg weight differed greatly between the dwarfs and normals. The studies of Hagger and Marguerat (1985) suggested that better efficiency in layers might be obtained by selection for a lower yolk:albumen ratio, but the improvement would be very small. There was a tendency for lines selected for increased body fat to have increased output of egg fat (Leclercq et al., 1985). The low yolk cholesterol lines selected by Marks and Washburn (1977) had a higher percentage yolk than did the high cholesterol lines. The Egyptian Fayoumi and Danclarami breeds and the native Iraqi breed, selected under a hot environment, had higher percentage yolk and lower percentage albumen than did imported breeds such as Rhode Island Red and White Leghorn (Hafez et al., 1955; Amer, 1972; Al-Rawi and Amer, 1972).

The percent albumen and percentage albumen solids of brown egg strains was significantly greater and the percentage yolk significantly less than that of the white egg strains in the study of Curtis et al. (1985).

Somes et al. (1977) obtained a higher yolk:albumen ratio in blue shelled Araucana eggs than from larger sized brown or white shelled eggs which did not differ in the ratio.

Calculated on an equal egg weight basis, the Araucana eggs had 23 percent more yolk and 9 percent less albumen than the White Leghorn eggs but weighed 87 percent of the White Leghorn eggs (Simmons and Somes, 1982). When comparisons were made of blue and white shelled eggs with a common genetic background (Sadjadi et al., 1983) there was no effect of the blue shell allele (*O*) on egg components suggesting the background genome may have influenced comparisons of the effect of the *O* gene.

Several studies have investigated the association of percentage of component parts and traits of economic importance such as egg production, body weight and hatchability. Rodda et al. (1977) found some evidence of genetic correlation between yolk percentage solids and age at first egg (-.48), body weight (.25), and egg production (.15 to .33). Correlations with albumen percentage solids were low for all the production traits. Phenotypic correlations for yolk weight and yolk solids with egg numbers were very low (.08 to .11) although the corresponding genetic correlations were high and negative (-.76 to -.38). A similar difference in phenotypic and genotypic correlations also occurred for albumen (Hill et al., 1966). Within the same commercial size category, commercial layers or strains selected for egg production had less yolk and more albumen and albumen percentage solids than did the nonselected control strain (Akbar et al., 1983).

Variation in egg weight and in the proportion of yolk and albumen have been associated with hatchability (Godfrey, 1936; Scott and Warren, 1941; Strong and Nestor, 1980). These studies have shown that large egg size with its greater percentage albumen is not compatible with high hatchability. This relationship is usually curvilinear but Strong and Nestor (1980) reported a linear relationship in a strain of turkeys selected for large body weight.

Effect of age. As the hen ages, the percentage yolk increases and the percentage albumen decreases (Olsson, 1936; Cunningham et al., 1960; Hafez et al., 1955; Fletcher et al., 1981). These changes in percentage yolk and albumen also affect the percent solids content of the egg (Rose et al., 1966). The genetic variation involved in the associated physiological changes with aging and changes in proportions of egg components may not be consistently expressed. Jull (1924) concluded that the component parts of the egg contribute differently to egg weight at different times of the year. Hill et al. (1966) reported a significant sire x age interaction for egg weight and albumen weight and in the study of Marion et al. (1964), in which percentage yolk and percentage albumen differences were noted for lines differing in egg weight, the magnitude of the difference changed somewhat with age.

Other species. Mahmoud and Coleman (1967) found that bobwhite quail eggs had less percentage albumen and more percentage yolk than did Japanese quail (40.8 vs. 47.4 albumen and 39.8 vs. 31.9 yolk). Ricklefs and Marks (1983) reported a range of 31.5 to 36.8 for percentage yolk and 43.6 to 56.1 for percentage albumen in lines of Japanese quail differing in body weight. The percentage yolk was greater and percentage albumen less in the smaller eggs from the nonselected line than in the eggs of the selected lines. Nestor et al. (1982) reported that the percentage yolk of turkeys ranged from 30.5 to 32.8 and percentage albumen from 57.5 to 59.7.

Selection studies and approach. The studies which have examined genetic variability within populations are meagre and selection experiments on proportions of egg components have not been conducted. Scheinberg et al. (1953) reported h^2 estimates of percentage albumen of .68 in New Hampshire, .60 in White Leghorn and .12 in Barred

Plymouth Rocks. Yolk h^2 was low for all breeds and the genetic variation was primarily additive. Hill et al. (1966) gave h^2 estimates of .49 for yolk percentage solids and .72 for albumen percentage solids. Jaffé (1964) obtained a value of .43 for yolk weight. Yao and Skinner (1959) reported a value of .30 for percentage albumen and .26 for percentage yolk.

Breeding programs that concentrate on egg production and egg size may result in changes in proportions of component parts of the egg. Although there does not appear to be a direct effect as the egg size increases, several studies have shown that the percentage yolk decreases and the percentage albumen increases as the egg size increases. Hill et al. (1966) concluded that the percent solids trait was biologically independent of egg weight and it did not have a negative genetic correlation with egg numbers. Thus, they suggested that simultaneous selection for egg weight, yolk percentage solids, and albumen percentage solids might produce the most rapid change of the egg component. Rose et al. (1966) concluded that selection of strains laying large eggs with high solids content could be achieved by selecting for a high yield of yolk (percentage yolk) since the yolk size was the primary controller of solids content of eggs obtained within an age category. Since the egg composition of two-way crosses was very predictable from the values of their parents, Abplanalp et al. (1984) suggested that egg composition traits could be modified by using appropriate parent lines.

ALBUMEN QUALITY

Albumen quality is an important egg characteristic because the proportions of firm albumen are used as a criterion for egg quality; consumers prefer eggs with a larger proportion of firm albumen. Albumen consists primarily of water and protein. Variation in albumen quality results from variation in quantity, quality, or arrangement of the protein portion.

Although albumen height and albumen score are sometimes used to measure albumen quality, critical reviews have concluded that Haugh units (log of albumen height corrected for egg weight) give the most satisfactory estimate.

Genetic basis. A number of studies have compared the albumen quality of various breeds, the objective of some of these being to show differences between brown and white shelled eggs. In pure breeds shell color and breed differences are confounded, thus comparisons between commercial strains may be more valid since they usually are a composite of more than one breed.

Most comparisons have shown that the Rhode Island Red breed and other brown egg strains including commercial layers have better albumen quality than the Leghorn breed or other white egg strains (Knox and Godfrey, 1934; Nordskog and Cotterill, 1953; Rodda, 1972; Farnsworth and Nordskog, 1955; Curtis et al., 1985). In addition, Mountney and Vanderzant (1957) reported differences between inbred strains of White Leghorns.

The h^2 estimates for albumen quality range from very low and negative to very high. Scheinberg et al. (1953) reported .66 in White Leghorn and .12 in Barred Plymouth Rock breeds, Nordskog and Farnsworth (1953) reported .22 to .28 for sire family estimates and .11 to .52 for dam family estimates, Farnsworth and Nordskog (1955) reported -.12 to .36 with an overall estimate of .22, Johnson and Merritt (1955) reported .55 for White Leghorns and .17 for Barred Plymouth Rocks, King et al. (1961) reported -.13 to .40 for fresh and -.02 to .49 for stored eggs, Redman and Shoffner (1961) reported 0 to .19, King (1961) reported .10, Hicks et al. (1961) reported -.13 to .26, and Poggenpoel (1986) reported .51 for sire component estimates and .28 for dam component estimates.

Although the estimate of .38 averaged over a number of studies by Van Tijen and Kuit (1970) indicates that selection for albumen quality would be effective, the variability in the estimates obtained indicates that selection would not be straightforward and suggests caution. Most of the variation in these studies could be attributed to differences obtained at different periods of measurement within the same flock and differences between brown and white shelled breeds.

Redman and Shoffner (1961) estimated that general combining ability accounted for over 60 percent of the genetic variation in albumen quality and dominance variation was 15 times greater than additive variation. Evidence for sex-linkage for genes involving albumen quality was observed and it was suggested by Poggenpoel (1986) that the sire may have more influence than the dam on albumen quality.

The best evidence for genetic variation in albumen quality is obtained from selection studies utilizing Haugh units as a measure of albumen quality. Lorenz and Taylor (1940) were able to select for significant divergence in high and low percentage of firm albumen. From these studies they concluded that genetics exerts some control over egg white. In the five-year selection in a Rhode Island Red population by Knox and Godfrey (1940) the percentage thick albumen of the high line increased from 50 to 69 percent and decreased to 47 percent in the low line. Albumen height was increased from 5.2 mm to 5.8 mm while Haugh units increased from 74.3 to 79.2 in six years of selection in a White Leghorn population (Poggenpoel, 1986).

Effects of nongenetic factors. Several nongenetic factors have been shown to affect albumen quality, including age of hen, length of storage, and season. These environmental factors become important since they interact with genetic variation to influence accuracy of measurement and selection progress.

Numerous studies have shown that Haugh units decrease with the age of the hen (Nagai and Gowe, 1969a; Orr, 1973; Rodda, 1972). The data of Strain and Johnson (1957) and Mueller (1959) indicated that the total phenotypic variability in albumen quality of a flock increases with age. The h^2 for albumen quality was shown by Poggenpoel (1986), Nagai and Gowe (1969b) and Rodda (1972) to decrease with age, suggesting that a greater accuracy of selection would be obtained by measuring albumen quality early in the laying year. However, most studies indicate a high correlation between values obtained early and late in the laying year (Poggenpoel, 1986; Nagai and Gowe, 1969a; Rodda, 1972) and that an accurate estimate of breeding value can be obtained from part-year records.

Doyon et al. (1986) found that although there were significant differences between commercial Leghorn strains and changes with age, the interaction of strain and age accounted for a very small amount of the variation associated with age. Several studies suggest an interaction between population differences and age differences. Johnson and Merritt (1955) reported that although the albumen quality of the White Leghorn was initially higher, it declined with age at a more rapid rate than did that of the Barred Plymouth Rock breed. The results of May et al. (1957) indicated differences between strains in the rate of albumen quality decline with age.

Albumen quality begins to decline soon after the egg is laid. Genetic variation in the rate of this decline in quality is an important consideration. Several studies have concluded that there was no measurable effect of genetic variation on the rate of loss in albumen quality. A genetic correlation between values of fresh and stored eggs of .94 to .99 was obtained by McClary and Bearse (1956). The h^2 estimates of stored or fresh eggs were shown by Poggenpoel (1986) to be similar and the genetic correlation between fresh and stored eggs was high (.86). In contrast, May et al. (1957) found significant differences among strains in rapidity of decline in albumen quality after storage and reported a relatively lower (.36) correlation between initial score and score after storage. In one randombred population the h^2 of albumen quality loss over a two-week storage period was .25 (sire component) or .57 (dam component) (Kyle and Mitchell, 1958), while the h^2 of Haugh unit loss in another randombred population ranged from .20 to .43 (King et al., 1961).

The actual effects of genetic variation in albumen quality loss in storage may be difficult to separate from genetic variation in initial albumen quality. The initial Haugh unit score was reported by Mueller (1959) to be responsible for about 66 percent of the variation in quality loss. The genetic correlation of -.88 obtained by King et al. (1961) between the value for Haugh unit loss and the value for percent stored of fresh suggests that the genetic variation in percent loss of quality is similar to that for the initial value.

Association with other traits. In a selection program to improve albumen quality, changes in other traits of economic importance are of prime concern, particularly egg production and egg weight. Knox and Godfrey (1938) found no relationship between egg production and albumen quality measured as percentage of firm white. Jeffrey (1941) noted a trend for hens with lower egg production to have higher albumen quality. There was a low but significant negative correlation between albumen quality and egg production in pedigreed populations at one location, but not at another, in the study of Brandt et al. (1953). A low but significant negative phenotypic correlation between albumen height and egg production was also observed by Johnson and Merritt (1955) in both White Leghorn (-.25) and Barred Plymouth Rock (-.18) breeds. Genetic correlations between albumen height and egg production were -.65 and .11 in the White Leghorn and Barred Plymouth Rock populations. However, the numbers of individuals and sire families were probably not sufficient for a reliable test of genetic variance.

A phenotypic correlation of -.3 between percent production and Haugh units was reported by King (1961), but such extreme fluctuations were observed over time that reliable estimates of genetic correlations were not obtained. Pope et al. (1960) did not observe a relationship between albumen quality and egg production over shorter periods.

Van Tijen and Kuit (1970) summarized the genetic correlations between egg production and albumen quality. The mean value obtained from several studies was -.08. In addition, Emsley et al. (1977) reported values of -.2 to -.3 for the correlations between egg production and albumen quality and Poggenpoel (1986) found low (-.20) correlations between production and albumen quality in a closed Leghorn population. A number of studies (Knox and Godfrey, 1934, 1938; Wilhelm and Heiman, 1938; Yao, 1958; Bornstein and Lipstein, 1962; Noles and Tindell, 1967; Van Tijen and Kuit, 1970; Curtis et al., 1985) have noted a relatively small relationship between albumen quality and egg size.

In selection for albumen quality there are concerns as to how soon after onset of lay measurements can be taken, how many eggs are needed to obtain a reliable estimate, and the effect of season. A number of studies have shown a seasonal effect on albumen quality (Lorenz and Almquist, 1936; Hunter et al., 1936; Knox and Godfrey, 1938). Thus, in selection programs and comparisons between groups, the time frame in which the data are collected becomes important. Several studies have indicated that genetic differences in Haugh units were maximized at certain periods (Hicks et al., 1961, December-January period; Raffa, 1963, first four months of the random sample tests). Heritability estimates were greatest at the earlier stages of the laying year (Nagai and Gowe, 1969b; Hicks et al., 1961; Johnson and Merritt, 1955). Repeatability estimates for Haugh units are generally high (Nordskog and Cotterill, 1953, .75; Farnsworth and Nordskog, 1955, .42; Nagai and

Gowe, 1969b, .7 to .8). These results suggest that pullets should be selected for albumen quality on the basis of early records using 4-5 eggs per pullet.

CHOLESTEROL

Because of the evidence which correlates plasma cholesterol levels with coronary heart disease it is recommended that the dietary cholesterol intake be restricted to less than 300 mg per day. If this recommendation is followed it would have a severe negative impact on egg consumption. Although it may be possible to reduce the cholesterol level of the hen's egg by dietary methods, these are not consistent, and may result in other problems. Thus, a genetic approach to permanent reduction in egg cholesterol would be desirable.

Genetic variation. Variation between breeds and strains in yolk cholesterol has been reported. Cholesterol (mg per g fat) ranged from 39.2 to 50.6 with highly significant differences between eight strains representative of the chickens available at that time (Edwards et al., 1960). Collins et al. (1968) found that two Leghorn stocks had significantly lower levels of yolk cholesterol than three heavy breed stocks. Cholesterol content was shown by Sáinz et al. (1983) to be similar for White Leghorn, Rhode Island Red, Sussex, and Maran breeds, but was lower for the English Gamecock breed. The mg cholesterol per g yolk of White Leghorn and Australorp commercial strains ranged from 12.5 to 13.9 with significant differences between strains (Sheridan et al., 1982).

The yolk cholesterol level of the meat-type Athens-Canadian randombred population was higher than that of the White Leghorn randombred population (Washburn and Marks, 1985; Marks and Washburn, 1977). There were significant sire effects on yolk cholesterol level in the randombred populations and h^2 estimates of .2 and .3 were obtained (Washburn and Nix, 1974). Subsequently, Marks and Washburn (1977) selected lines for divergence in yolk cholesterol concentration in these populations. The high and low lines differed significantly in both populations after selection (1.27 mg and 1.38 mg). However, in both populations the yolk cholesterol in the low line was not significantly different from that of the nonselected control population. Realized h^2 estimates ranged from .11 to .25 in the Athens-Canadian population and .21 to .25 in the Leghorn population. The lack of selection response in the low line suggested that selection for yolk cholesterol was effective only in the upward direction in this population.

Cunningham et al. (1974) reported the yolk cholesterol in a closed White Leghorn population ranged from 921 mg percent to 1756 mg percent. The heritability based on intrasire regression of offspring on dam was .24 and realized h^2 based on response to one generation of bidirectional selection was .21. A significant difference of 71.4 mg percent cholesterol was obtained after one generation of bidirectional selection. However, there were no nonselected controls for comparisons.

Becker et al. (1977) reported a h^2 of .19 and .15 for mg cholesterol per g of yolk in eggs from a randombred White Leghorn population, obtained from sire variance components and regression, while realized h^2 ranged from .04 to .13. Based on the lack of significant differences between sires and significance between dams only at P=.1, Sadjadi et al. (1983) concluded there was little evidence of genetic variation for yolk cholesterol in their population.

In contrast to the study of Marks and Washburn (1977), Ansah et al. (1985), using an index of individual and sib records, were able to decrease genetically the yolk cholesterol in a randombred Leghorn population by 5.4 percent in three generations. The cholesterol for the low line after three generations was 13.9 mg compared to 14.7 mg in the control line. A realized h^2 of .26 was obtained.

Reports in the popular literature (Hickman, 1974) have indicated that the cholesterol level of blue shelled eggs (due to the *O* gene) is lower than that of brown or white shelled eggs. However, comparative scientific studies have not substantiated this fact. The cholesterol content of blue shelled eggs from three strains of Araucanas was consistently higher (2-6.9 percent) than that of white or brown shelled eggs (Somes et al., 1977). The cholesterol content of Araucana egg yolks averaged 4.0 percent higher than that of white shelled eggs while that of brown shelled eggs was intermediate in the study by Cunningham (1977). In a cross designed to provide full sisters that were *O/O* or *O/o*, Sadjadi et al. (1983) found no differences in the yolk cholesterol of white and blue shelled eggs. Eggs from Araucanas had significantly more total cholesterol (27 percent) and yolk cholesterol (7 percent) than eggs from White Leghorns in later studies by Simmons and Somes (1985).

Association with other traits. In the study of Edwards et al. (1960), there was an inverse relationship between percent egg production of the strains and their egg yolk cholesterol level. Harris and Wilcox (1963) found a negative correlation of -.17 with egg production and -.14 with body weight, neither of which was statistically significant. Collins et al. (1968) found inconsistent correlations between egg production and egg cholesterol ranging from -.54 in one stock to +.19 in another. Significant negative correlations of yolk cholesterol and egg production of low magnitude (-.14 and -.15) were reported by Washburn and Nix (1974). Cunningham et al. (1974) reported correlations of -.27 and -.38 between yolk cholesterol level and egg production in a closed Leghorn population.

Selection for divergence in yolk cholesterol also resulted in divergence of percent egg production in both populations used by Washburn and Marks (1977). Percent egg production was 8.0 percent greater in the low line than in the high line. The phenotypic correlations averaged over selection generations were -.17 and -.20 in the high and low Leghorn lines and -.12 and -.04 in the high and low Athens-Canadian lines. Ansah et al. (1985) found correlations between egg production and yolk cholesterol to be near zero in three years of testing, but in one year there were significant negative correlations (-.18 and -.27). Turk and Barnett (1971) reported that egg production strains had a lower yolk cholesterol content than broiler strains. Bartov et al. (1971) found that cholesterol concentration per unit of yolk was significantly higher in eggs laid by poor producers.

In the study of Edwards et al. (1960) there was a trend for the heavier birds to lay eggs with a higher cholesterol level (r=.44) and with more fat (r=.26). The egg and yolk weights of lines selected for high yolk cholesterol by Marks and Washburn (1977) were significantly higher than that of lines selected for low yolk cholesterol in both a Leghorn and a meat-type population. However, phenotypic correlations within lines and populations were low (.04 to .10). This difference in egg weight was associated with the differences in adult body weight between the lines. Washburn and Marks (1985) confirmed the higher egg weights in the high line but correlations of yolk cholesterol and egg weight were low in both populations ($\bar{x}$ .05). Ansah et al. (1985) found no correlation between yolk cholesterol and egg weight in either their control or lines selected for yolk cholesterol.

The requirement of the embryo for cholesterol (Andrews et al., 1968) would suggest that attempts to lower the egg cholesterol past a certain level would result in reduced hatchability. However, in a randombred population not selected for cholesterol level there was no correlation between yolk cholesterol level and hatchability (Washburn and Marks, 1977). Cunningham et al. (1974) found a positive correlation of .25 for hatchability and yolk cholesterol in a closed Leghorn population. Hatchability of the low yolk cholesterol group was 78.6 compared to 89.1 percent in the high cholesterol group.

Selection for three generations in a Leghorn population and four generations in a meat-type population for high and low yolk cholesterol by Marks and Washburn (1977) did not result in a decrease in hatchability of the low cholesterol line and the phenotypic correlations were low (.05 and -.04).

Differences among various studies in the association between yolk cholesterol levels and hatchability may be related to the level of hatchability and fitness of the populations and may vary from year to year in the same population. In some studies, which used closed populations with low hatchability, an association was shown (Cunningham et al., 1974), while in other studies using randombred populations with higher hatchability (Washburn and Nix, 1974) there was no association.

A number of studies such as those of Harris and Wilcox (1963), Bartov et al. (1971), Washburn and Nix (1974), and Marks and Washburn (1977), have noted two aspects concerning variability in yolk cholesterol of various populations of hens. First, although yolk cholesterol in different eggs from the same hen in a yearly period are quite consistent, there is wide variation between individual hens. If this individual variation is nongenetic it could interfere with selection progress. Secondly, values for yolk cholesterol from the same population using the same techniques and observers are quite variable between years, emphasizing the need for adequate control populations.

Methodology. The estimated cholesterol content of 274 mg per egg (Consumer and Food Economics Institute of U.S.D.A.) and most of the values obtained in genetic studies were primarily based on data obtained by the Zlatkis et al. (1953) colorimetric determinations. Recent work by Beyer (1988) has shown that these determinations are subject to interference from noncholesterol substances, resulting in an overestimation of yolk cholesterol compared to determination by high performance liquid chromatography (HPLC). Since it would be extremely difficult to obtain the numbers necessary for genetic studies by using the HPLC method, further studies are necessary to determine the extent of correlation between values obtained by more rapid colorimetric methods and HPLC methods. If there is a high correlation between values obtained by the two methods, the more rapid but overestimating Zlatkis method may be of more value in genetic studies.

FATTY ACIDS

Indirect evidence of differences among breeds and strains in fatty acid composition of the hen's egg was obtained by Edwards et al. (1960). The iodine number, an indication of the unsaturation of fat, was different among the various strains tested. There appeared to be no relationship of iodine number of the egg fat to class of bird or shell color. Further studies by Edwards (1964) found significant differences between strains in the content of palmitoleic acid, stearic acid, linoleic acid, and arachidonic acid. Sell et al. (1968) found differences among strains in fatty acid composition of yolk and ova, but these differences were small in relation to differences resulting from variation in dietary fat. Farr et al. (1972) compared the fatty acid composition of egg yolk of two strains of bobwhite quail; they differed significantly in stearic and oleic acid.

Ferreira et al. (1976) fed diets containing different energy levels to commercial egg laying strains. The levels of oleic acid, stearic acid, and palmitoleic acid differed between strains and there was an interaction between strains in the effect of energy levels on fatty acid concentration. Eggs from lines selected for low body fat (Leclercq et al., 1985) had a different fatty acid profile than eggs from high fat lines. The low fat line had less myristic and palmitoleic acid, but more stearic acid.

Variations in the fatty acid content of the yolk could contribute to variation in hatchability and growth since Menge et al. (1964) demonstrated that essential fatty acid depletion of dams resulted in reduction of the arachidonic and linoleic acid content of their progeny which hatched later and grew slower than progeny from hens not depleted.

Marion et al. (1966) found that the levels of fatty acids showed significant age x line effects which would indicate that the effects of age differ among genetic groups.

Genetic differences in some chromatographic fractions of the yolk (mostly phospholipids) were shown by Seideman and Cotterill (1969). Variation of this type may be responsible for the breed differences in emulsifying capacity of yolk observed between breeds by Varadarajulu and Cunningham (1972).

VITAMINS

Riboflavin. Maw (1954), in studying the causes of poor hatchability in a flock of White Leghorns, noted that their albumen lacked the greenish-yellow color of normal eggs. This was shown to be due to the absence of riboflavin in these eggs even though the diet contained adequate riboflavin. The condition was the result of a recessive gene, assigned the symbol *rd,* which results in embryonic death by riboflavin deficiency.

Numerous studies have been conducted to elucidate the physiological and biochemical mechanism of this genetic lesion. Boucher et al. (1959) concluded that the gene altered the metabolism of the egg laying female in such a way that the normal increase in free riboflavin of the blood did not occur. A riboflavin-binding protein, induced by blood plasma estrogen levels, is present in the blood of normal females in egg production. This protein in a functional form is not present in *rd/rd* females (Farrell et al., 1970). Although riboflavin is intestinally absorbed from the diet of the *rd/rd* individual, it is not transported to the developing yolk nor does it become incorporated into the albumen deposited during egg formation.

There is some evidence of a connection between riboflavin deficiency and clubbed down and poor hatchability. However, the dead *rd/rd* embryos were shown not to have clubbed down (Hawes and Buss, 1965). In addition to the differences in riboflavin content associated with the *rd* gene, Collins et al. (1970) reported variation in the yolk riboflavin content of hens segregating for *B* vs. *b* and *K* vs. *k* genes.

Thiamine. Howes and Hutt (1956), Scrimshaw et al. (1945), and Mayfield et al. (1955) found that the White Leghorn breed (13 strains) used thiamine more efficiently than heavy breeds (13 strains representing four breeds) and that they deposited more of the dietary thiamine in their eggs. However, Arroyave et al. (1957) did not find a similar difference. In the study by Scrimshaw et al. (1945) there were no significant differences within the White Leghorn or heavy breeds but there were significant differences between sire families in the thiamine content of eggs from a Barred Plymouth Rock x White Leghorn cross.

MINERALS

Bolton (1957) found that various inbred Brown Leghorn lines differed in their ability to transfer manganese from the diet to the egg resulting in symptoms similar to that obtained when fed manganese deficient diets.

The sex-linked gene *ro* described by Jones et al. (1975) results in a condition described as restricted ovulator. Hens with the sex-linked restricted ovulator gene lay a few small-sized eggs that contain yolks that are translucent and deep orange in color resulting from a failure to incorporate the normal amounts and proportions of low density lipoproteins and lipovitellin (Schjeide et al., 1976). Egg yolks from hens with the *ro* gene contained less copper and iron but more sodium and potassium than eggs from normal hens (Grau et al., 1979). In addition, when the yolk was subdivided into supernatant and granule fraction, the granule fraction of the restricted ovulation eggs contained less manganese and the supernatant contained more potassium, magnesium, and zinc than eggs from normal hens.

Simmons and Somes (1985) found that the yolks of Araucana eggs had seven percent more calcium, ten percent more zinc, and three percent more iron than White Leghorn eggs. Arroyave et al. (1957) reported significant differences between breeds of chickens for egg content of nitrogen, phosphorus, vitamin A, and carotene. However, no single breed was consistently superior in all nutrients tested.

ABNORMAL METABOLIC PRODUCTS (TAINTED EGGS)

Fishy taints in eggs are a dramatic example of the importance of normal metabolism in the hen. Vondell (1932) reported a situation in which some individuals in a flock of Rhode Island Reds produced eggs with a fishy odor and taste. Subsequent studies (Vondell, 1948) indicated this was primarily a problem of brown egg layers (Rhode Island Red and Barred Plymouth Rock) and they only produced the tainted eggs when fed a diet containing rapeseed meal.

A number of other studies (Hobson-Frohock et al., 1973, 1975; Miller et al., 1972; Blair et al., 1975; Hawrysh et al., 1975) have also shown similar types of variation between breeds and in brown egg commercial layers. Variation in individual expression within a flock and interaction with diets were also shown by these studies. These findings suggested a genetic basis interacting with dietary nutrients to produce tainted eggs.

Studies by Hobson-Frohock et al. (1973) demonstrated that the tainting substance was trimethylamine (TMA). Although there is very little TMA in rapeseed meal, the studies of Hobson-Frohock et al. (1975) and Hawrysh et al. (1975) confirmed that the tainted eggs could be produced by feeding rapeseed meal to susceptible flocks. It can also be produced by feeding fish meal (Fenwick et al., 1981).

The studies of Bolton et al. (1976) demonstrated that tainting is dependent on the presence of an autosomal semidominant mutant gene that has a variable expression, depending on environmental factors. Studies by Pearson et al. (1979) indicated that the deficiency was highly heritable in both males and females. Bolton et al. (1976) suggested that the tendency towards tainting of eggs in birds fed rapeseed meal is not associated with the production of brown shelled eggs, but it is associated with the ancestry of the birds used to produce brown shelled eggs. It was originally thought that the deficiency was associated with brown shells since the earlier comparisons were mostly with Rhode Island Reds which lay brown eggs and White Leghorns which lay white eggs. In addition, studies by Griffiths et al. (1980) showed that there is a very low incidence of deficiency in TMA oxidase activities in broiler stocks which lay brown eggs. The low incidence may reflect introduction of the Rhode Island Red genome into the population at some previous time. Although the presence or absence of rapeseed in the diet is a primary dietary factor influencing the production of tainted eggs, it is not the only one since the incidence in any individual bird is variable.

The genetic deficiency is expressed as a deficiency of TMA oxidase (Pearson et al., 1979). Studies by Butler et al. (1980, 1982) also demonstrated that TMA oxidase is the site of the genetic deficiency. TMA in the normal hen is oxidized to triethylamine oxide. If there is a deficiency of TMA oxidase, the hen is unable to metabolize all of the TMA derived from the diet. The TMA is excreted in the egg resulting in a tainted egg. The proportion of tainted eggs produced by susceptible stock apparently depends on the interaction between the ability of the bird to produce the TMA oxidase which is genetically controlled as well as the concentration of TMA or TMA precursors in the diet.

MEAT AND BLOOD SPOTS

A number of studies have shown variation between breeds in the incidence of meat and blood spots. No significant breed differences in blood spots were found by Van Wagenen and Wilgus (1935), but those laying brown shelled eggs had a higher frequency of meat spots. Brown shelled eggs from Barred Plymouth Rocks and Rhode Island Reds were shown by Hall (1939) to have a higher incidence of meat spots than the white shelled eggs of White Leghorns, but White Wyandottes which also laid brown shelled eggs had an incidence similar to that of White Leghorns. Jeffrey (1945) also found that meat spots were very common in brown eggs, had a lower frequency in tinted eggs, and were seldom seen in white eggs. A generally higher incidence of blood spots and lower incidence of meat spots in white shelled eggs from White Leghorns than in brown eggs were reported by Dawson et al. (1954) and King and Hall (1955). Eggs from the Fayoumi breed contained a higher percentage of meat and blood spots than Rhode Island Reds and White Leghorns (Amer, 1961). A protoporphyrin mutant studied by Shoffner et al. (1982) drastically reduced egg shell pigment. This was associated with a visible reduction in meat spots compared to brown and tinted eggs.

The incidence of blood and meat spots may increase with age and these changes with age may not be consistent for strains differing in incidence (Strain and Johnson, 1957).

Jeffrey and Pino (1943) and Lerner and Smith (1942) concluded from their studies that the incidence of blood spots had a genetic basis. Six generations of selection for high or low incidence of blood spots by Quinn et al. (1948) resulted in lines with 80 and 20 percent incidence. Although the incidence of blood spots had a high h^2 (.5) and could easily be influenced by selection (Lerner et al., 1951), it was not possible to completely eliminate it. Much lower h^2 estimates were obtained by Johnson (1956); these were .21 and .07 for blood spots in White Leghorns and Barred Plymouth Rocks and .25 for meat spots in Barred Plymouth Rocks.

PROTEIN CONTENT

Although there is a large amount of variation among hens, variation among breeds in total egg protein is small (Cotterill and Winter, 1954). Small but significant differences in nitrogen content were found among five breeds examined by Arroyave et al. (1957). The strain of hen may influence egg percentage protein (May and Stadelman, 1960). The age of hen and egg size may also affect egg percentage protein. Since there is a high correlation between weight of egg contents and percentage protein, the variation among strains in percentage protein may be related to variation in egg weight.

The protein content of blue shelled Araucana eggs was shown by Somes et al. (1977) to be lower than those of White Leghorns or Sex-links. This was due to the increase in the yolk:albumen ratio of the Araucana eggs. However, Cunningham (1977) using electrophoretic studies found no differences in kind or amount of protein in albumen of eggs produced by Araucana, White Leghorn, or Plymouth Rock hens. Eggs from the English Gamecock contained a higher proportion of protein than other breeds (Sáinz et al., 1983).

ENERGY

Sibbald (1979) reported gross energy per gram of egg ranged from 1.33 to 1.47 kcal and differed significantly among three strains of White Leghorn. The strains differed in egg size and the variation in energy was associated with this variation in egg size. The gross energy per g values for turkey and goose eggs were similar, but slightly greater than for hen eggs.

YOLK DISORDERS

Jeffrey (1945) observed that the Barred Plymouth Rock and Rhode Island Red breeds were more prone to have defective yolks than were other breeds. Farnsworth and Nordskog (1955) estimated that the h^2 of yolk color is 0.15. King et al. (1961) reported a very high h^2 (.73) based on sire and dam components for mottling score of fresh eggs and a high h^2 (.53) estimate after storage. Renden et al. (1984) found that percent yolk mottling was lower in eggs from hens selected for low body weight compared to those selected for high body weight.

VARIATION IN SHELL CHARACTERISTICS

The shell of an egg consists of the cuticle (bloom), the calcium carbonate portion of the shell, and the shell membranes. Characteristics of the shell of importance include the appearance of the cuticle, shell strength, color, porosity and mottling, shell texture, and the thickness and composition of the shell membranes. This discussion will encompass inherent genetic variation in these characteristics and how they might relate to other characteristics. Results of selection for these characteristics are included in Chapter 29.

Shell strength. It is estimated that the shells of 6-8 percent of all eggs are damaged before the eggs can be used by the consumer (Washburn, 1982). Egg shell cracks and breakage are influenced by a combination of genetic, physiological, and environmental variables, most of which influence shell damage by affecting shell strength. Shell strength can be assessed by a number of methods which are related to the qualitative or quantitative composition of the shell.

Variation in shell strength between individuals and between various populations has been observed. A number of these comparisons have focused on the differences in shell strength of brown and white shelled eggs. The percent shell of brown shelled eggs from the Barred Plymouth Rock breed was shown by Taylor and Martin (1928) to be lower than that of white shelled eggs from the White Leghorn breed. Munro (1938) found significant differences among dam families in percent shell ash, leading to the conclusion of significant differences in shell strength. Taylor and Lerner (1941) demonstrated genetic variation in the shell thickness within a White Leghorn population. Significant differences in shell thickness between various breeds and lines were observed in the studies of Farnsworth and Nordskog (1955) as follows: Leghorn .30 mm, Fayoumi .35 mm, New Hampshire .31 mm, Rhode Island Red .30 mm, Barred Plymouth Rock .29 mm, Australorp .28 mm, and Langshan .30 mm. Tyler and Geake (1958) have also shown that the eggs from White Leghorns had thicker shells than eggs from the Rhode Island Red breed. Their data suggest that the superiority of shell strength in white eggs obtained from the White Leghorn breed may not be associated solely with shell color, since thickness of white shelled eggs from the Brown Leghorn breed was similar to that of brown shelled eggs from the Rhode Island Red breed. Nagai and Gowe (1969b) reported significant differences in egg specific gravity for three strains of White Leghorns. Perek and Snapir (1970) also presented data showing differences in shell strength of brown and white shelled eggs; the mg per cm of shell was greater in White Leghorns than in White Rocks which lay brown shelled eggs. Carter (1970, 1971) found significant differences between several strains in tensile strength. A Rhode Island Red strain had poorer shell strength assessed by shell thickness or specific gravity than the White Leghorn strain used by Rodda (1972). Significant differences were observed in percent broken eggs in lines of White Rocks selected for divergence in body weight (Siegel et al., 1978).

In addition to the differences observed between lines and breeds selected for different specific traits and characteristics, there is also evidence for differences in shell strength of breeds selected under different environments. Arad and Marder (1982) found that a desert inhabiting breed (Sinai) had stronger and thicker shells than did commercial White Leghorns. The differences observed in egg size and egg mass of the two breeds did not explain the differences in egg shell quality. The porosity of the Sinai breed was less which, combined with the greater thickness of the shell, could be considered an adaptation to the arid environment under which the Sinai breed was developed.

Comparisons of shell strength have been made among different lines of commercial layers. Bowman and Challender (1963) found significant differences among eight commercial strain crosses in shell breaking point, thickness, and percent cracks. Potts and Washburn (1974) compared the shell strength of three white and three brown egg commercial strains. Significant differences were found among the strains. The shell strength of the white egg strains was superior to that of the brown egg strains. The decrease in shell strength with age began earlier and decreased at a faster rate in the brown egg strains. Hamilton et al. (1979a) compared the shell strength (percent shell, shell weight, specific gravity) of ten commercial strains and strain crosses and the changes in shell strength with age. Differences were observed between the strains in shell strength measures and in the relative changes with age. Hunton (1982) summarized shell strength (specific gravity) of the commercial strains in random sample tests from 1963 to 1979. Overall, the eggs from the white egg strains had superior shell strength compared to the brown shell stocks.

Factors which introduce variation in shell strength within a species (shell thickness, shape, size, deformation) account for less than 60 percent of the variation in shell strength (Carter, 1971; Voisey and Hamilton, 1976; Hunt et al., 1977). The sources of naturally occurring variation in shell strength of five domestic avian species (Japanese quail, turkey, duck, goose, chicken) were studied by Thompson et al. (1981). Within species, their study agrees with previous work assessing the source of variation in shell strength. However, the factors introducing variation in shell strength across species were not necessarily the same as those within species.

Potts and Washburn (1974) found that the relationship between specific gravity and nondestructive deformation or shell breaking strength were all influenced by the strain of commercial layer used. Hamilton et al. (1979b) reported that the strain from which eggs were obtained significantly affected the values for nondestructive deformation and compression fracture force.

The h^2 estimates calculated for shell strength are variable. The h^2 estimates in the seven breeds studied over a three-year period by Farnsworth and Nordskog (1955) ranged from .09 to .24. Johnson and Merritt (1955) reported h^2 estimates for specific gravity of .32 in White Leghorns and .56 in Barred Plymouth Rocks. The h^2 estimates decreased over the ten-month period from .44 to .21 in the Leghorn population but were similar for the Barred Plymouth Rocks over this period. Jaffé (1966) reported sire and dam h^2 estimates for specific gravity in a White Leghorn population to range from .28 to .47. Heritability estimates obtained over a three-year period and in three strains by Nagai and Gowe (1969b) ranged from .22 to .44. Carter (1971) found that the incidence of shell cracking, although only slightly affected by thickness at oviposition, was heritable and should respond rapidly to selection for decreased incidence.

Heritability estimates for a Rhode Island Red strain ranged from .57 to .33 when the birds were 33-65 weeks of age, while estimates for a Leghorn strain ranged from .27 to .05 over this period (Rodda, 1972). The h^2 estimates for specific gravity in White Leghorn strains ranged from .49 to .57 over a four-year period. The h^2 estimates in both populations decreased with age. Potts and Washburn (1985) reported sire family h^2 estimates for shell deformation in a commercial Leghorn parent line of .30 from 1-8 weeks of lay, .38 from 9-17 weeks, and .18 at 21 weeks. The h^2 of frequency of cracked eggs in a White Leghorn population ranged from .11 to .43 (Engström et al., 1986).

Shell color. The color of the egg shell in chickens usually varies from white to a dark brown, but it may be blue or greenish. The white/brown shell color is inherited as a multifactoral trait and may be influenced by modifying genes (Benjamin, 1920; Punnett and Bailey, 1920). On the basis of crossbreeding experiments, several studies (Punnett and Bailey, 1920; Hall, 1944) concluded there were sex-linked genes affecting shell color. The intensity of shell color in matings between white and brown shelled populations is intermediate between the shell color of the parent stock (Hall, 1944). The intensity of the brown shell color can be quite variable within breeds or strains as well as between breeding groups, requiring as many as 13 shades to describe it (Kopec, 1926).

The h^2 of shell color in brown egg strains is high. Blow et al. (1950) reported a range of .30 for sire component estimates to .91 for dam component estimates. Variation in shell color in a number of lines was evaluated by Farnsworth and Nordskog (1955). Significant differences were observed between lines, and h^2 estimates of .21 to .46 were obtained for shell color. The h^2 of shell color in a Light Sussex flock ranged from .34 to .45 for sire and dam component estimates. The estimate obtained from the dam component always exceeded the estimate obtained from the sire, suggesting dominance effects. Jaffé (1966) reported a h^2 estimate of shell color of .34-.54 in a White Leghorn population, .47 in a Light Sussex population, and .70 in a Rhode Island Red population.

The blue or green shell color is due to the presence of an autosomal dominant gene (*O*) first described by Punnett (1933). The *O* gene on the multigenic background for white shell color results in a blue shell color while the *O* gene on the multigenic background for brown shell color results in a greenish (olive drab) shell color.

The pigment responsible for the color in brown shelled eggs is a hemoglobin porphyrin (protoporphyrin IX). The major pigments responsible for the blue or green shell color are biliverdin, a zinc chelate of biliverdin, and the protoporphyrin IX found in brown shells.

Shoffner et al. (1982) reported a recessive sex-linked mutant (*pr*) which results in a lack of protoporphyrin pigment in the shell from hens with the polygenic brown shell color. Although this gene has the potential of removing undesirable tints from the eggs of white shelled strains, it is also associated with decreased egg numbers and size. The *pr* gene is closely associated with a translocation between chromosome 1 and the Z sex chromosome (Schwartz et al., 1980).

There is disagreement concerning the association of shell color with other shell characteristics. Godfrey (1949) found significant correlations in the New Hampshire breed between tint and specific gravity (.39), between tint and breaking strength (.36), and between tint and shell thickness (.36). Grover et al. (1980) found a small (+.17) but significant correlation between shell color and specific gravity in a commercial brown egg strain. Carter (1975) concluded that shell color had a great influence on the incidence of cracks and other shell defects. The darker brown eggs of the Vasca breed were stronger (.25 correlation) and thicker (.21 correlation) than those with lighter color (Campo and Escudero, 1984). In Japanese quail, white shelled eggs were not as strong as those of normally pigmented eggs (Briggs and Williams, 1975).

In contrast, Briggs and Teulings (1974) using the Athens-Canadian randombred population, and Shoffner et al. (1982) comparing eggs of the Rhode Island Red, found no association of shell color and shell strength. Potts and Washburn (1974) using a stepwise regression technique found that the tint of brown shelled eggs from commercial layer lines did not contribute significantly to either nondestructive deformation or breaking strength. Potts and Washburn (1974) found very low correlation between intensity of shell color and shell strength assessed by several methods in another study using commercial layers.

In other traits, Rhode Island Red lines selected for high egg numbers showed a correlated decrease in intensity of brown shell color while shell color of a line selected from the same base population for large egg size was not affected (Hunton, 1964). Lines selected over 22 generations for growth differed in shell thickness and color with the high line having darker shell color and thicker shells (Goodman and Shealey, 1977).

Cuticle. Both the amount and distribution of the cuticle may be variable. A chalky type was shown to be inherited as a dominant character which can be modified, resulting in an intermediate condition (Taylor and Lerner, 1941).

Shell membrane. Studies on shell quality have focused primarily on the crystalline portion of the shell. Electron microscopy has demonstrated the importance of the shell membrane as the calcification site on which the crystalline structure is laid down (Parsons, 1982). The studies of Britton (1977) indicated that eggs with poorer shell strength had less shell membrane by weight and per gram of egg. Brown shelled eggs which also had weaker shells tended to have lower membrane weights than white shelled eggs. Simmons and Somes (1982) reported that the membrane weights of eggs from a White Leghorn strain were heavier than those of blue shelled eggs from the Araucana breed. The absolute and relative weight of the shell membrane was shown by Asmundson (1939) to vary between avian species. Turkey eggs had a heavier shell membrane than eggs from chickens.

Shell texture. Farnsworth and Nordskog (1955) reported significant differences between breeds and lines in shell texture. Heritability estimates ranged from -.40 to +.48. The texture of the shell, particularly the sandiness sometimes observed, appears to be inherited by recessive genes (Hays, 1937). Shell mottling has been shown to have a high repeatability within strains and differences between strains have been shown (Baker and Curtiss, 1958).

Shell porosity. Porosity is an important contributor to the quality and hatchability of the egg since it influences the passage of gases as well as microorganisms through the shell. Almquist and Hoist (1931) demonstrated a direct relationship between the number of pores and weight loss of the egg during storage. They observed uniformity of shell porosity for individual hens but differences between hens, suggesting the possibility of genetic variation. Variation between breeds in weight loss during storage was shown by Godfrey and Olsen (1937); the eggs from Rhode Island Reds showed significantly more weight loss than those of White Leghorns. Quinn et al. (1945) were able to select divergent lines for weight loss during storage. An association was shown between the egg weight loss during storage and thinness and degree of brittleness as well as porosity. Mountney and Vanderzant (1957) found that Leghorns laid eggs with more porosity than those of two inbred lines.

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Chapter 33. DISEASE GENETICS

J.S. Gavora

Survival and good health of poultry are fundamental for efficient production. Genetically based natural resistance to disease has been recognized for a long time as an important factor in disease control. This chapter deals with the following aspects of such resistance: types and definitions of health problems; evolution of disease resistance; mechanisms of resistance and resistance to specific diseases; and genetic improvement of resistance, including molecular genetic approaches.

Global estimates of losses from disease in poultry are unavailable, but the available data sufficiently justify continuing efforts for their reduction. Damage caused by poultry diseases can result from the following: mortality; morbidity; negative effects of subclinical infections; costs of vaccination, medication, and other veterinary care; reduced genetic progress from selection; and deterioration of human health from zoonoses and cost of laboratory safety precautions.

TYPES OF HEALTH PROBLEMS, DEFINITION OF DISEASE RESISTANCE AND ITS TYPES

Poultry health problems can be subdivided into nutritional disorders, stress, developmental disorders, and infectious diseases. Nutritional diseases include deficiencies or excesses of nutrients, consequences of nutritional imbalances, or effects of poisonous feed ingredients. Genetic variation in the ability of birds to cope with these problems is not well understood, but it likely exists since strains 'resistant' to deficiency of an amino acid (argine or lysine) have been developed by genetic selection (Hutt and Nesheim, 1966; Godfrey, 1968). Nevertheless, it is unlikely that significant efforts will develop to produce commercial poultry resistant to nutritional problems.

Stress results from exposure to a hostile environment (Scott, 1981). The severity of stress is, at least partly, genetically determined. Selection experiments show that stress syndrome in chickens and turkeys can be genetically modified (Brown and Nestor, 1970, 1973, 1974; Gross and Colmano, 1971; Dickerson and Mather, 1976; Hansen, 1976; Gross et al., 1984; Gross and Siegel, 1985). Stress is known to influence the levels of some hormones that can also affect immune response and thus influence disease resistance. Plasma corticosterone levels are increased in stressful situations. Low levels of corticosterone are not favorable for resistance to bacterial infections but are associated with increased resistance to viral diseases and mycoplasmoses (Gross and Colmano, 1971). A line of chickens selected for high plasma corticosterone levels after social stress showed an impaired cell-mediated immune response and increased susceptibility to Marek's disease compared to a low plasma corticosterone-selected line (Thompson et al., 1980).

Developmental disorders include genetic, morphological, and physiological problems caused by deleterious genes. The disorders can behave as quantitative characters or could result from a single gene. Since major gene effects are covered in other parts of this book, this chapter discusses only disorders of a quantitative nature.

Infectious diseases are a major concern for the poultry industry. There are, in principle, two types of resistance: resistance to infection and resistance to disease development.

Resistance to infection, or true resistance, reduces or prevents infection. Complete resistance to infection is rare and is exemplified by lymphoid leukosis. It is usually specific for a pathogen and is inherited in a Mendelian fashion. Partial resistance to infection is brought about mainly through mechanisms of body surface protection. Resistance to disease development, or disease tolerance, is usually polygenically inherited, and it is less specific with regard to the type of pathogen than resistance to infection. It is the most common type of disease resistance.

From the practical breeders' point of view it would be desirable to identify general disease resistance mechanisms effective against a multiplicity of diseases (Gavora and Spencer, 1978) but there is little evidence that such resistance, usable in practical breeding, exists.

A third and often forgotten type is nonhost resistance in which a pathogen is unable to infect or induce disease in species other than its usual host. For example, plant viruses are unable to infect animals. The model for evolutionary behavior of nonhost resistance should be of interest to poultry geneticists (Lundgren et al., 1982) because its understanding can be useful in efforts to improve resistance to poultry diseases.

EVOLUTION OF RESISTANCE

Parasitism in the broad sense is so widespread that every organism is either a parasite or a host or both. Therefore, a general understanding of co-evolution of hosts and parasites should aid in planning optimal breeding strategy for disease resistance.

Viral and bacterial diseases tend to become less virulent through time (Day, 1974). This is caused by group selection because highly virulent parasites could become extinct when they destroy their host. The evolutionary objective of both the host and the parasite is its survival as a species. The objective of the host-parasite interaction appears to be the development of a balance, leading to the survival of the system (Gavora and Spencer, 1983). As resistance of the host increases, for instance by vaccination or genetic means, the host can tolerate more virulent pathogens. This may be the reason for the appearance of highly virulent strains of Marek's disease virus (Witter, 1988).

The immune system had a major role in the evolution of resistance. Biozzi et al. (1979), based on research in mice, suggest that the immune system evolved to adapt the host defence against diversified and increasingly sophisticated aggression by parasites. They consider phagocytic cells to represent the first evolutionary step that is nonspecific and lacks memory. The second step is cell-mediated immunity, and the final one is the ability to synthesize and release highly stereospecific molecules, the antibodies. Cell-mediated and humoral immune systems contain lymphoid memory cells and take advantage of the individual's immunological history. Biozzi et al. (1979) claim that the above three immune functions are under separate polygenic control. Results of Van der Zijpp (1983) and Lamont and Smyth (1984a, 1984b) from experiments in chickens are in agreement with this suggestion.

Before domestication, selection for survival and resistance to disease was taking place. Such selection continues in some breeds that are not subject to vaccination and selection programs. After domestication, when sophisticated methods of protection against pathogens were introduced, natural selection for survival and disease resistance was dramatically reduced. Artificial selection concentrated on improvement of production traits with little attention to resistance to disease. Nevertheless, in the selection for production, providing the birds were exposed to disease, they were also being selected for resistance since only survivors could be used for breeding. However, such selection was often erratic as there was little control over the exposure of breeding flocks to pathogens.

RESISTANCE MECHANISMS

Disease resistance mechanisms can be broadly classified into two categories: immunological, and nonimmunological. Because avian immunology is covered in Chapter 22 of this book, this chapter only deals with involvement of immune mechanisms in disease resistance. Nonimmunological mechanisms will be described in more detail.

Immunological Mechanisms

Cellular immune response. Available data on avian macrophages suggest that, as in mammals, they are essential as cells that initiate antibody production, in addition to being phagocytic (Sharma and Tizard, 1984). Both functions are essential in resistance to diseases.

Genetic differences in T-cell response have been demonstrated. T-cell antigens Ly-4 and Th-1 are associated with resistance to Marek's disease and Rous sarcoma (Gilmour et al., 1983). Infection of chickens from resistant line 6 and susceptible line 7 with Marek's disease virus resulted in depressed response to phytohemaglutinin, an indicator of T-cell activity (Lee et al., 1981). Natural killer (NK) cells do not clearly fit into the classical immune scheme, since effector NK cells are present prior to exposure to antigens against which they are reactive, and they are also present in SPF chicks (Sharma and Coulson, 1979; Sharma and Tizard, 1984). Tumor target cells vary in susceptibility to NK cells (Sharma and Okazaki, 1981). NK cells may also play a role in genetic or vaccine-induced Marek's disease resistance (Sharma, 1981) and can transfer resistance to chickens against transplantable tumors (Lam and Linna, 1979, 1980).

Graft-versus-host reaction (GVHR) indicates cell-mediated immune response. A line of chickens, homozygous for the B^{11} MHC haplotype and selected for high GVHR, was more susceptible to Marek's disease than its low GVHR selected counterpart. No such difference was seen in a similar pair of lines homozygous for B^{9} (Ashikaga et al., 1984; Okada and Yamamoto, 1987; Yamamoto et al., 1988).

Humoral immune response. A line of chickens selected for high antibody titres to sheep red blood cells showed stronger antibody response to Newcastle disease virus, and was more resistant to *Mycoplasma gallisepticum*, *Eimeria necatrix*, a splenomegaly virus, and feather mites, but less resistant to *Escherichia coli* and *Staphyloccocus aureus* than a line selected for low titres (Siegel and Gross, 1980; Gross et al., 1980). A line selected for persistence of antibody response was more resistant to all infectious agents tested than that selected for nonpersistence (Gross et al., 1980). Divergent selection for antibody response to Newcastle disease virus in Japanese quail significantly separated high and low response lines (heritability .12 ± .50), but no difference was observed in their survival (Takahashi et al., 1984).

Genetic control of humoral response and susceptibility to Marek's disease in chickens were associated with response to a simple amino acid polymer GAT; hence gene(s) involved in Marek's disease resistance map within the major histocompatibility system (Pevzner et al., 1981a, 1981b; Steadham et al., 1987). A line of chickens selected for low IgG levels in serum was more resistant to Marek's disease than a corresponding line selected for high IgG. However, lines selected for high and low antibody response to rabbit serum albumen or for high and low anaphylactic shock response did not differ in resistance to Marek's disease (Yamamoto et al., 1988).

A possible association of immunoglobulin levels with the phenotype of inherited muscular dystrophy in chickens was demonstrated by Abplanalp and Benedict (1978). Correlations among antibody responses to a variety of antigens in chickens were suggested by the study of Balcarová et al. (1973). Hence, it may be possible to select for a general intensity of antibody formation.

Major histocompatibility complex. The major histocompatibility complex (MHC) plays an important role in resistance to disease. (For a detailed discussion of the MHC see Chapter 22). It is the B-F region of the MHC that appears to be responsible for resistance to Marek's disease (Briles et al., 1983). The precise regions related to resistance will likely be more evident as the MHC genes become molecularly defined (Xu et al., 1988; Guillemot et al., 1988).

Studies on the association of MHC with resistance to disease have involved mostly White Leghorns. Preliminary evaluation indicates existence of a much wider spectrum of allomorphs in other breeds (Simonsen et al., 1982a). Therefore, further 'resistant haplotypes' can be expected when more MHC research is extended to other breeds. It should be also kept in mind that, with the possible exception of B^{21} and MD resistance, all other alleles identified as favorable were found to be so in particular populations and it is still uncertain whether a specific B-haplotype can be superior in all strains (Bacon, 1987). The mechanisms through which the MHC influences resistance to disease are not clearly understood. However, evidence is accumulating that the products of the B-system are important in the presentation and processing of antigenic determinants of pathogens during immune response.

The association of the MHC with disease resistance is summarized in Table 33.1. There is strong and consistent evidence for the superiority of the B^{21} allele over other alleles with regard to resistance to Marek's disease. In a study combining selection for high egg production, related traits, and disease resistance, haplotypes B^{21} and B^{2} were dramatically increased in the resistance-selected and production-selected strains (Gavora et al., 1986). Differences in frequencies of MHC haplotypes were also found to be associated with feed efficiency and egg production (Lamont et al., 1987b). Selection response of MHC haplotype frequencies may depend on environment and exposure to pathogens. In a Scandinavian selection study, B^{15} was favored in flocks selected for egg production in cages, but decreased in those selected on the floor (Simonsen et al., 1982b).

B^{15} is shown mostly as a 'susceptible' allele in Table 33.1. Therefore, it is surprising that B^{15} is homozygous in the Cornell K strain developed by selection for resistance to disease in general, including MD, and high egg production and egg weight (Cole, 1985). Perhaps B^{21} was absent from the base population for strain K. Also, the USDA RPRL lines 6 and 7 differ widely in MD resistance but both are B^{2} homozygous, indicating that multiple mechanisms must be involved in MD resistance (Pazderka et al., 1975).

Diseases other than MD show involvement of the MHC. Reduced lymphoid leukosis virus shedding by chickens with certain MHC haplotypes was reported by Bacon (1987). Similarly, involvement of the MHC in coccidiosis, infectious bursal disease and fowl cholera may prove important. The suggestion by Bacon (1987) that poultry breeding companies should seek information about their primary breeding stock with regard to the MHC, needs to be re-emphasized.

Table 33.1. Association of major histocompatibility (*B*) system haplotypes with disease resistance. (Modified from Bacon, 1987).

Disease, test conditions, chickens[1]	*B* haplotypes[2]	Reference
Marek's disease		
Exposure to macerated tissue or field exposure: Hy-Line hybrids	19; 21	Hansen et al. (1967)
Field exposure: DeKalb hybrids	2; 6; 13; 19	Briles and Oleson (1971) Briles and Briles (1982)
JM-MDV[3] at 2 weeks : JM-NxJM-P crosses	13; 19; 21	Briles and Stone (1975) Briles et al. (1977)
Exposure to BC-1-MDV shedders: ED1, ED2 (F_2 hybrids)	2; 14; 21	Longenecker et al. (1976)
BC-1-MDV homogenate at 3 weeks: Ottawa lines XP, XR, XN	2, 21, X*	
JM-MDV at 2 weeks: Regional Cornell Randombred	13; 15; 5; 19; 21	Briles et al. (1976) Briles et al. (1980a)
HPRS-16-MDV at 3 weeks: commercial cross	2/21; 5/21; X*/21; 15/21; 19/21	Briles et al. (1980b)
JM-MDV at 2 weeks: 6_3x15_1 F_5 cross	2; 5	Bacon et al. (1981b)
JM-MDV at 2 weeks: DeKalb hybrids and recombinant cross	21/19; 19/19; F21-G19/19	Briles et al. (1983)
JM-10, GA-5, or RB-1B-MDV at 2 days: UCD-003 inbred and 7 *B*-congenic lines	2; 15; 17; 18; 19; 21; Q*; 24	Abplanalp et al. (1985)
Exposure to MDV shedders at 1-14 days: several lines varying in MD resistance	No correlation of *B*-type with resistance	Van dem Hagen and Löliger (1985)
BC-1 MDV at 1 day: Ottawa strains 7,8,9 and 8R selected for high egg production and MD resistance	(f) of 21 and 2 increased, 13, 12 and Y retained, decreased or eliminated with the selection	Gavora et al. (1986)
JM-MDV:$15I_5$ B-congenic	21; 5; 2; 15; 12; 13; 19	Bacon (1987)
Transient paralysis		
GA-MDV at 3 to 4 weeks and paralysis after 8 days: G-B1 x G-B2 hybrids	13, 6	Schierman and Fletcher (1980)
Sarcoma tumors		
BH-RSV[4] (RAV-1) at 6 weeks in wing-web: 6_1 x 15_1 F_2 hybrids	2; 5	Collins et al. (1977)

Continued ...

Table 33.1. Association of major histocompatibility (*B*) system haplotypes with disease resistance. (Modified from Bacon, 1987). (Continued)

Disease, test conditions, chickens[1]	*B* haplotypes[2]	Reference
Schmidt-Rupin RSV at 4 weeks in wing-web: G-B1 x G-B2 hybrids	13; 6	Schierman et al. (1977) Schierman and Collins (1987)
BH-RSV (RAV-1) at 6 weeks in wing-web: UNH 105 New Hampshires	22; 24; 26	Collins et al. (1985)
Erythroblastosis		
Avian leukosis virus (RAV-1) at 1 week: 6_3 x 15_1 F_5 hybrids	2; 5	Bacon et al. (1981b)
Lymphoid leukosis		
(RAV-1) at 1 week: 6_3 x 15_1 F_5 hybrids	2; 5	Bacon et al. (1981b)
Field exposure: Ottawa strain 7	2; 12; 13; 15; 19; 21 (reduced virus shedding)	Bacon (1987)
Spontaneous autoimmune thyroiditis		
Thyroid pathology and antibody to thyroglobulin: obese strain (OS) inbreds (Cornell)	15; 13; 5	Bacon et al. (1974)
OS x CS F_2 (Cornell)	6; 15	Bacon et al. (1981a)
Coccidiosis		
$15I_5$ *B*-congenic lines exposed to *Eimeria tenella* or *E. ascervulina*	2; 5; 12; 13; 15; 19	Ruff and Bacon (1984)
Auburn line A selected for resistance (R) and susceptibility (S) to *E. tenella*	Three alleles in moderate (f) in S line absent in R line. R line has 2 high (f) alleles, one absent in S, the other lower (f) in S.	Johnson and Edgar (1986)
6_1 x 15_1 F_4 challenged with *E. tenella;* cecal lesions and delayed wattle reaction	2/2; 2/5; 5/5	Clare et al. (1985)
Infectious bursal disease		
IBDV vaccine; development of neutralizing antibody; *B*-congenic lines	2; 5; 12; 13; 15; 19; 21	Bacon et al. (1987)
Fowl cholera		
P. multocida strain X73 at 3 weeks: Iowa State line S1	1, 19	Lamont et al. (1987a)

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[1] White Leghorns unless otherwise specified.

[2] Standard nomenclature of Briles et al. (1982) except where asterisk indicates a tentative symbol. The haplotypes with a solid underline indicate relatively resistant (regressor if sarcoma), and with no underline, susceptible genotypes; (f)=gene frequency.

[3] MDV=Marek's disease virus.

[4] RSV=Rous sarcoma virus.

Nonimmunological Mechanisms

Most of the nonimmunological mechanisms protect the surface of the bird's body and tissues against attachment and penetration of pathogens and can be classified as: *physical*, that include desiccation, pH extremes, desquamation, mucus barriers, fluid flows (saliva, vomiting, diarrhea, coughing), and body temperature; *chemical*, such as fatty acids, gastric acid, and proteolytic enzymes; and *biological*, including pathogen receptors, competition with normal microflora, and antibiosis. Of these, only a few have been studied in poultry but it can be assumed that genetic factors play an important role in the establishment and effectiveness of the mechanisms.

Hutt and Crawford (1960) found that selection for rapid attainment of higher body temperature in chicks was effective in reducing mortality from *Salmonella pullorum*. Blood pressure has been shown to affect mortality in chickens, with hypotensive bird mortality being double that of groups with higher blood pressure (Sturkie et al., 1956). Selection changed blood pressure levels of chickens, but no differences in mortality of the high and low blood pressure lines were observed (Sturkie, 1970). These controversial results may relate to the presence of different pathogens in the populations studied.

A chemical protection mechanism, lysozyme, is an antibacterial and antiviral enzyme synthesized in many tissues and present in most body fluids, being highest in tears and egg white. Wilcox and Cole (1957) reported establishment by selection of high and low egg white lysozyme strains, and suggested that the strain differences may be due to modifying genes or the presence of multiple copies of the lysozyme gene. Almost 30 years later it was shown that regulation of lysozyme production indeed involves a unique feature: the presence of an enhancer, a DNA sequence 6.1 kb upstream to the 5' transcription start of the gene (Thiesen et al., 1986). The enhancer distinguishes the lysozyme gene from other genes such as the insulin and chymotrypsin genes (Walker et al., 1983; Edlund et al., 1985), where the tissue-specific control elements are located directly in the 5' noncoding region of the gene. The lack of mortality or production trait differences between the lines selected by Wilcox and Cole (1957) should not be interpreted as indicating that lysozyme is not important. Perhaps the changes by selection in the levels of lysozyme in egg white, present at extremely high levels to start with (Wilcox and Daniel, 1954), did not have any significant consequences in the birds' physiological state.

The interferon system, another important nonimmune resistance mechanism, is involved in resistance against viral diseases. Stimulation of production of interferon in chicken trachea and kidney cells by chemical interferon inducers increases resistance of these tissues to several viruses (Finkelstein, 1972). The relatively Marek's disease resistant Cornell strain K was reported to produce a higher level of interferon compared to a related susceptible strain S (Hong and Sevoian, 1971). However, Kaleta (1977) concluded, on the basis of a review of literature and his own experimentation, that interferon is not involved in Marek's disease resistance. Surprisingly, none of six strains of the avian infectious bronchitis virus was found to be susceptible to chicken interferon (Holmes and Darbyshire, 1978).

Lack of receptor substances on host cells that facilitate the entry of pathogens is an important defense mechanism. The most information is available on the lymphoid leukosis virus receptor system. The absence of suitable receptors renders chickens resistant to the virus. Pathogens opportunistically utilize existing substances on the host cells as their receptors. Such substances may have a physiological function for the host and, therefore, be genetically stable. Thus it is not surprising that resistance to infection due to absence of suitable receptors is rare, and avian leukosis is the only known example of such a situation in birds. Through studies of receptors for these viruses, an understanding may be gained of reasons for their structural variation that makes some of them inaccessible to the virus.

In diseases involving neoplastic transformation there seem to be mechanisms, which are as yet little understood, that determine the resistance of the target cells to transformation. The bursa of Fabricius is the source of target cells for the neoplastic transformation by lymphoid leukosis viruses (Purchase and Gilmour, 1975). Postinfection genetic resistance to lymphoid leukosis resides at least partly in the B-target cell (Purchase et al., 1977). In Marek's disease, the target cell is a T-lymphocyte which also has a role in immune surveillance against the disease (Sharma et al., 1977). All chickens are susceptible to infection with Marek's disease virus. Resistance to transformation by the virus was demonstrated at the level of the target cell, and it was suggested that resistant birds may have fewer receptors, receptors of lower affinity for the virus, or fewer target cells with identical receptors (Gallatin and Longenecker, 1979).

Presence of low molecular weight neutralizing substance(s) in a line of chickens resistant to Rous sarcoma virus (regressors), but not in susceptible progressor chickens, was reported by Whitfill et al. (1983).

Age And Disease Resistance

Age plays a significant role in resistance to some diseases. Onset of immune competence in chickens can be modified by selection (Ubosi et al., 1985; Pitcovski et al., 1987). In meat-type chickens selected for high and low early response to *Escherichia coli* and Newcastle disease virus vaccine, the response was 68 percent higher in the high line than in the low line, with a realized family heritability of .67 to .72. Total mortality was 31 percent in the low and 11 percent in the high early response line, and the corresponding mortalities after *E. coli* challenge were seven percent and 23 percent (Pitcovski et al., 1987). This is in agreement with the observation that selection for low response to the synthetic antigen GAT increased mortality (Pevzner et al., 1981b).

Age resistance has been widely studied in Marek's disease. Cox (1971) indicated the existence of such resistance. However it is important to distinguish passive maternal antibody immunity from true age resistance. In antibody-free birds of the Athens-Canadian randombred control strain, Marek's disease mortality was 92, 92, 76, and 60 percent to 21 weeks after inoculation with MD virus at day 1, and 5, 10, and 15 weeks of age respectively (Anderson et al., 1971). Age at exposure influences early pathogenesis of Marek's disease (Calnek, 1972). Chickens of resistant lines can resist Marek's disease challenge even at hatching (Sharma, 1976).

The response of day-old chicks to challenge with the JMV-L tumor transplant was not influenced by maternal antibody, and both strain and age had pronounced effects on the lethal dose that would cause death of 50 percent of the populations (LD_{50}) (Table 33.2). Strains 4 and K were approaching complete resistance by the seventh day of age, but chickens of the susceptible strain S were more resistant by 14 days than the day-old chicks of strains 4 and K. Thus, early development of immunological competence may determine resistance to Marek's disease (Witter, 1976). In lymphoid leukosis, effects of age on resistance were documented by Maas et al. (1982).

It is generally assumed that immunological competence of birds declines with age starting from sexual maturity, but no studies on the relationship of older age immune response with genetic resistance to disease have been found. Suffice it to quote Hildemann (1981) who stipulates that "all major groups of multicellular animals, both invertebrate and vertebrate, possess immune recognition systems that defend somatic integrity but gradually allow immune dysfunction, a prime cause of aging".

GENETIC VARIATION IN RESISTANCE TO SPECIFIC DISEASES

From early investigations up to the present time, genetic variation has been almost without exception found whenever adequately searched for. An excellent review of the earlier work is provided by Hutt (1958).

Marek's Disease

Marek's disease is a lymphoproliferative disease of chickens caused by a herpesvirus. Genetic differences in Marek's disease resistance have been repeatedly demonstrated since the report by Asmundson and Biely (1932), and since Hutt and Cole (1947) showed that resistant or susceptible lines of chickens can be selected using natural exposure to the virus.

Significant effects of sire and dam on the susceptibility of progeny to Marek's disease in White Leghorn and Rhode Island Red chickens were shown by Biggs et al. (1968). In Columbian Plymouth Rock chickens selected for resistance and susceptibility to Marek's disease, a resistant line had fewer lesions in most tissues than a susceptible line, and nerve lesions were more frequent in males while gonads were more frequently involved in females (Morris et al., 1970). In 15 strains of chickens there were highly significant differences in MD resistance, and females were more susceptible than males (Grunder et al., 1972). They also found a significant correlation between the ranking of the strains on resistance to contact and injection exposure, thus providing the basis for further studies using the more convenient and uniform injection challenge route.

In a highly susceptible White Leghorn strain and a commercial meat-type strain exposed to acute or mild Marek's disease viruses, the meat-type birds developed viremia earlier and more often than the susceptible Leghorns. The mild virus viremia in the meat strain persisted longer. The appearance and persistence of viremia was similar in the two

Table 33.2. Effect of age and strain on resistance of chickens to the JMV-L tumor cell inoculum. (Modified from Gavora et al., 1977b).

Age at inoculation, days	Number of JMV-L cells that would kill 50 percent of the tested population (LD_{50})		
	Strain 4	Strain K	Strain S
1	10,480	691	18
7	AR (3/176)[1]	AR (4/176)	5,238
14	R (0/179)[1]	AR (1/178)	665,500
21	R (0/179)	R (0/175)	AR (4/100)

[1] AR=approaching complete resistance - low mortality observed was insufficient to estimate LD_{50}; R=completely resistant (number of birds dead/total number challenged).

strains after exposure to the acute virus (Cho and Kenzy, 1973). Similarly, a susceptible Leghorn line tended to show higher levels of viremia than a corresponding resistant line; overall levels of viremia were higher in both lines with an acute than with a mild virus (Cho, 1975).

MD incidence among 40 sire families from two strains of Leghorns and among 39 Leghorn inbred lines varied from zero to 46 percent, and the ranking of the sire families on MD resistance was not correlated with the ranking of their sisters on performance in egg production traits. There was no correlation between the coefficient of inbreeding and resistance to Marek's disease (Grunder et al., 1974). Flock et al. (1976) tested five commercial strains of egg laying chickens under contact and inoculation challenge with Marek's disease virus and presence or absence of Marek's disease vaccination. Significant strain differences were observed, but there was no strain x method of exposure interaction.

Testing for MD resistance under inoculation exposure requires an observation period of a minimum of eight weeks, and with contact exposure even longer. Therefore, alternative ways for determining resistance were sought. Gavora et al. (1977b) showed that resistance of chicks to the JMV tumor transplant was not influenced by maternal antibody, and that mortality to two weeks corresponded well to their known resistance to the Marek's disease virus. Attempts to use the response to *Listeria monocytogenes*, as a measure of cell-mediated immunocompetence and indicator of Marek's disease resistance, were not successful (Carpenter and Sevoian, 1982).

There were no striking differences between MD resistant and susceptible lines in precipitating antibody against MD virus but marked differences in virus neutralizing antibodies. Susceptible chickens developed severe lesions (gross and microscopic) by two weeks postinoculation, while resistant ones did not show any lesions up to 14 weeks after inoculation (Sharma and Stone, 1972).

In situ hybridization of two T-lymphoblastoid cell lines showed that Marek's disease virus DNA was integrated into the short arms of chromosomes 2 and 4 and was not in the vicinity of known oncogenes or integrated retroviral genomes (Hirai et al., 1986).

After the introduction of Marek's disease vaccination in the early 1970s, the interaction of genetic and vaccination-induced resistance became a prime interest. The relationship of vaccination and genetic resistance to Marek's diseases is illustrated in Figure 33.1. It was shown that vaccination reduces losses from Marek's disease but protection from vaccination is not complete, ranking of strains on Marek's disease resistance is not changed by vaccination, and both genetic and vaccination-induced resistance is necessary to maximize protection (Spencer et al., 1972, 1974; Gavora and Spencer, 1979).

The protection of nine strains of Leghorns by vaccination with cell-associated or lyophilized herpes virus of turkeys was compared by Gavora et al. (1977a). Both vaccines protected the chickens against a field exposure to Marek's disease virus, but the cell-associated resulted in lower total mortality and higher egg production than the lyophilized vaccine. Significant variation among the strains pointed out the dangers associated with drawing general conclusions about effectiveness of vaccines from testing only a small number of genetic stocks of chickens.

Of the genes controlling resistance to Marek's disease, the involvement of the MHC is the best documented. The B^{21} haplotype is clearly associated with Marek's disease resistance (Table 33.1). Other genes involved are immune genes regulating response to the synthetic polypeptide GAT (Pevzner et al., 1981b) and antigens Ly-1 and Th-4 (Gilmour et al., 1983) discussed above. The mechanisms through which these genes influence MD resistance are not clearly defined.

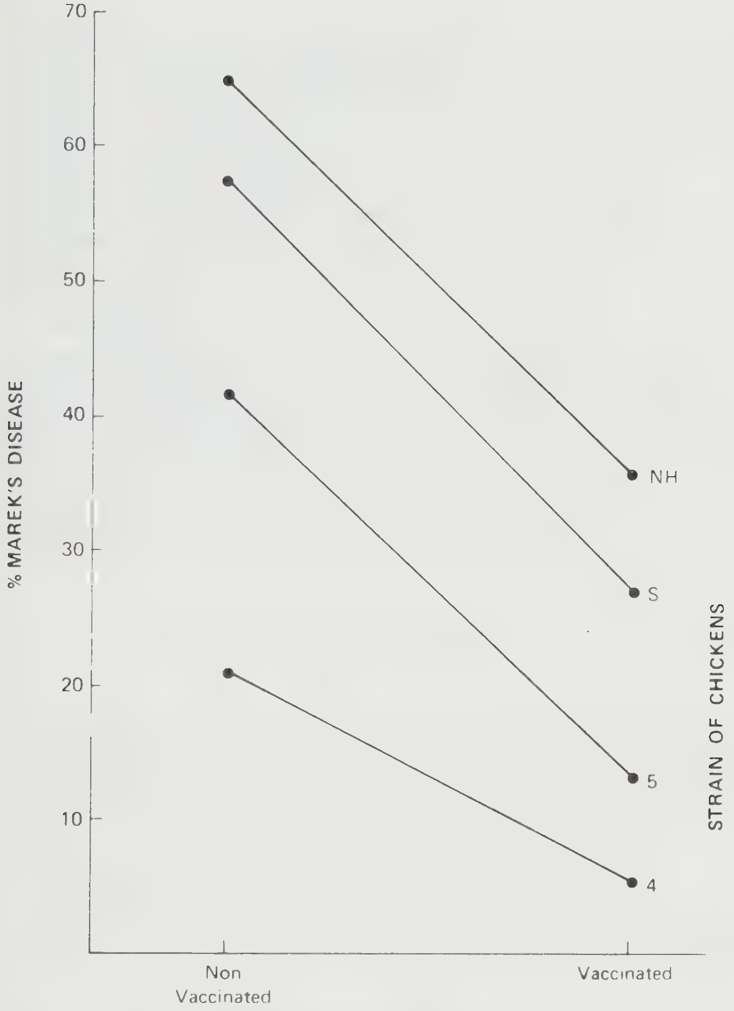

Figure 33.1. Response of genotypes to vaccination. Females exposed to Marek's disease by contact at three weeks. Based on lesions in birds that died or were killed at 133 days of age. (From Gavora and Spencer, 1979).

Lymphoid Leukosis

Of poultry diseases caused by retroviruses (RNA viruses), lymphoid leukosis (LL) has been the most widely studied. It will be discussed in detail, followed by a brief discussion of other retroviral diseases and oncogenes.

Avian lymphoid leukosis is induced by exogenous LL viruses of subgroups A, B, C, and D. The endogenous virus of the chicken belongs to subgroup E. Prior to efforts to eradicate exogenous LL viruses, nearly all flocks of chickens were infected by subgroup A LL virus (Crittenden, 1976).

Vertical transmission of LL viruses from a hen to her progeny and horizontal transmission from chicken to chicken has been recognized for some time (Burmester, 1957; Rubin et al., 1961; Crittenden, 1976). Early infection, either congenitally or through horizontal transmission of the virus, usually leads to the highest incidence of lymphoid leukosis. Congenitally infected chicks tend to develop a permanent viremia and are immunologically tolerant to the virus. Horizontally infected birds could become immune to the virus. Some infected hens transmit infection to their progeny in an erratic fashion (Rubin et al., 1962). Only dams and not sires transmit the LL virus to their progeny (Spencer et al., 1980; Gavora et al., 1983).

There are two levels of genetic resistance to LL (Crittenden, 1975). Cellular resistance to virus infection is the first level of resistance, or the first line of defence. Resistance to tumor development in infected birds constitutes the second level, or second line of defence.

Exogenous retroviruses replicate through a DNA intermediate integrated into the host cell DNA (Crittenden, 1981). In contrast, expression of the endogenous virus is controlled by DNA proviruses, or endogenous viral (*ev*) genes, permanently integrated into the host germ line and inherited in a Mendelian fashion. The structure of an avian retrovirus is shown in Figure 33.2. There are three viral genes or coding regions: *gag, pol, env* (Figure 33.3). In some viruses, such as the Rous sarcoma virus (RSV), an oncogene is also found in the viral genome. The viral genome and oncogenes are found in different combinations and reductions in the various avian retroviruses.

The penetration of the host cell by the virus (Figure 33.4) involves an interaction of the host cell with the viral envelope protein gp85 (Scheele and Hanafusa, 1971).

First line of defence. Three autosomal loci of the chicken control resistance to subgroups A, B, and C viruses (Table 33.3). Each can carry resistant or susceptible alleles. Loci *Tv-A* and *Tv-C* are closely linked. Multiple allelism at the *Tv-B* locus was described by Crittenden and Motta (1975), and one of the susceptibility alleles was shown to be closely associated with the R_1 blood group phenotype (Crittenden et al., 1970). As the R_1 antigen is not detectable on chick embryo fibroblasts which express the corresponding receptor, it is unlikely that the blood group antigen is identical with the virus receptor (Crittenden and Briles, 1971). The *Tv-B* locus also controls resistance to subgroup D virus. Genetic resistance to subgroup E virus is believed to involve two autosomal loci: *Tv-E* and an inhibitor locus *I-E* with a dominant resistant and recessive susceptible allele (Table 33.3).

Only the exogenous virus subgroups A and B are common in poultry flocks while endogenous proviral genes are present in almost all chickens. Most White Leghorn chickens are susceptible to infection with subgroups A and B, while resistance is more frequent in the meat-type and at a somewhat lower level in brown egg laying birds (Calnek, 1968; Crittenden and Motta, 1969; Spencer et al., 1979; Gavora et al., 1982; Crittenden et al., 1983b).

The low frequency of cellular resistance to infection in White Leghorns makes its use in practical breeding programs difficult. It appears more feasible to increase resistance in meat-type and brown egg stocks. Reasons for the difference in frequencies of cellular resistance to avian leukosis viruses between the meat-type and white egg-type chickens are not understood. They need to be examined in view of possible efforts to increase the frequency of this resistance in commercial stocks of chickens (Gavora, 1986).

Resistance or susceptibility to a leukosis virus infection can be determined by inoculating chick embryo fibroblasts in cell culture with Rous sarcoma virus of corresponding subgroup, that can cause neoplastic transformation observed as discreet pocks of tumor cells if susceptible (Temin and Rubin, 1958). Resistance can also be detected in cultured feather pulp cells (Crittenden et al., 1971; Payne et al., 1985; Spencer et al., 1987). Inoculation of RSV onto the chorioallantoic membrane (CAM) of 11-day-old embryonated eggs results, after another eight days of incubation, in the formation of tumors visible as pocks on the CAM (Dougherty et al., 1960). Lack of mortality or tumor incidence after subcutaneous or intramuscular (Greenwood et al., 1948; Burmester et al., 1960) or intracerebral inoculation (Waters and Fontes, 1960) with RSV can be also used as indicators of resistance to infection. Evidence that chickens resistant to a subgroup of RSV will be also resistant to the corresponding subgroup of LL viruses was reviewed by Payne (1985).

The cell receptor genes for the avian leukosis-sarcoma viruses have not been yet cloned and the structure of the receptors is not known. However, the *env* genes of all five subgroups have been cloned and sequenced. The predicted amino acid structure of the critical envelope proteins (Figure 33.2) from all five subgroups have been recently compared by Bova and Swanstrom (1987). There are 340 amino acids in the gp85 and there are four

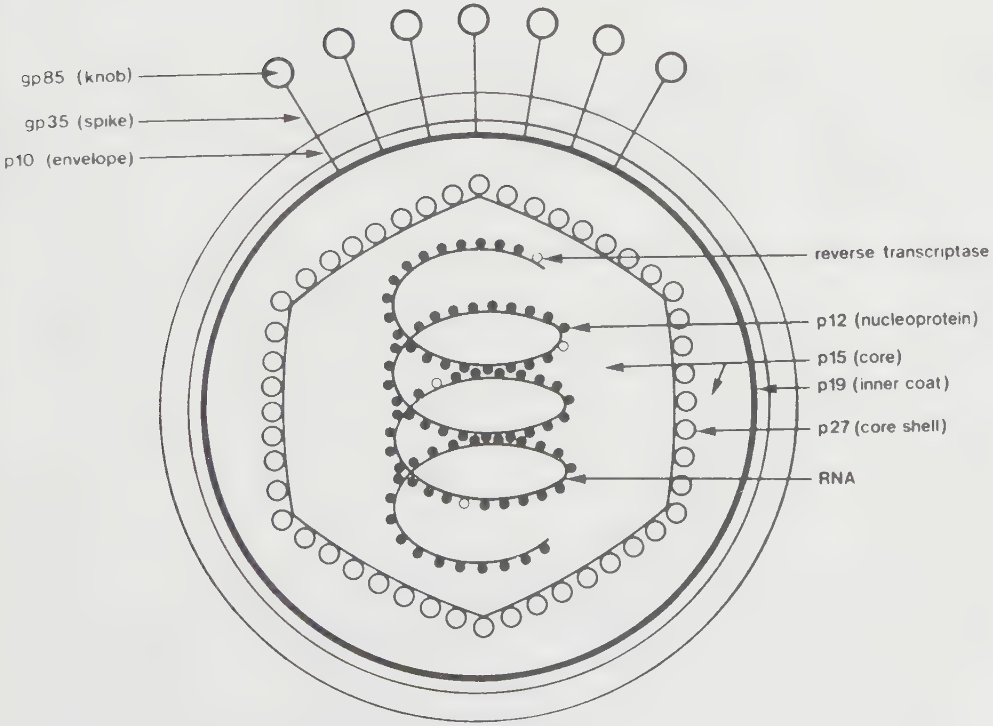

Figure 33.2. Diagram of an avian retrovirus. (From Payne, 1984).

Table 33.3. Genes of the chicken controlling cell susceptibility to infection with avian leukosis/sarcoma viruses. (Modified from Payne, 1985).

Virus subgroup	Locus	Alleles[1]	Reference
A	*Tv-A*	$Tv\text{-}A^{s}$, $tv\text{-}A^{r}$	Crittenden et al. (1964), Crittenden et al. (1967), Pani and Biggs (1973)
B and D	*Tv-B*	$Tv\text{-}B^{s1,s2,s3}$, $tv\text{-}B^{r}$	Rubin (1965), Payne and Biggs (1966), Crittenden et al. (1967), Crittenden and Motta (1975), Pani (1975), Pani (1976)
C	*Tv-C*	$Tv\text{-}C^{s}$, $tv\text{-}C^{r}$	Payne and Biggs (1970), Motta et al. (1973)
E	*Tv-E*	$Tv\text{-}E^{s}$, $tv\text{-}E^{r}$	Crittenden et al. (1973), Pani (1974), Crittenden and Motta (1975), Pani (1976)
...	*I-E*	*I-E*, *i-E*	Payne et al. (1971), Pani and Payne (1973), Pani (1977)

[1] Superscript s=susceptible (dominant), r=resistant; allele *I-E* is dominant at the *I-E* locus.

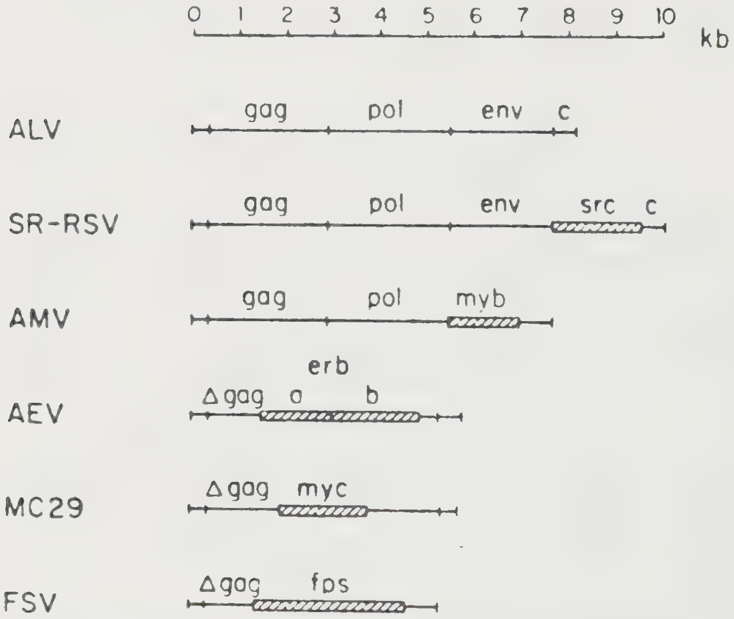

Figure 33.3. Genomic RNAs of the slowly transforming avian leukosis virus (ALV) and of representative rapidly transforming avian retroviruses: Schmidt-Ruppin strain of Rous sarcoma virus (SR-RSV), avian myeloblastosis virus (AMV), avian erythroblastosis virus (AEV), myelocytomatosis virus-29 (MC29), and Fujinami sarcoma virus (FSV). Viral genes encode the structural proteins (*gag*), reverse transcriptase (*pol*), and the envelope glycoproteins (*env*). The 'C' region includes sequences (U+R) that form the long terminal repeat (LTR) of proviral DNA, plus other sequences of unknown action. Oncogenes are shown by cross-hatched boxes, and are of cellular origin apparently acquired by recombination. (From Hayward et al., 1982).

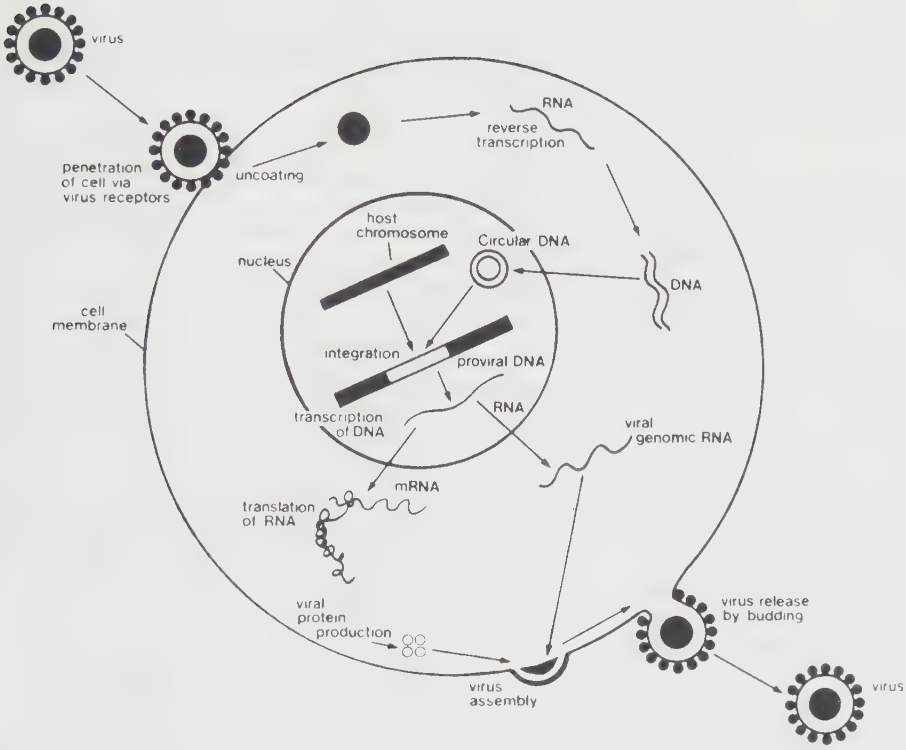

Figure 33.4. Major steps in the replication of an avian retrovirus. (From Payne, 1984).

variable regions (VR): VR-1, 12 amino acids; VR-2, 45 amino acids; VR-3, 229 amino acids; and VR-4, 9 amino acids. These regions are flanked by highly conserved sequences where, on an average, 95 percent of the amino acids are the same in all five subgroups.

Lines of Leghorn chickens resistant and susceptible to infection with subgroups A and B of avian leukosis viruses have been developed from three different genetic bases by selection based on tumor formation after inoculating RSV into the wing web (Hartman et al., 1984). The susceptible sublines showed higher total mortality during the laying period than the resistant lines, but the size of the differences was influenced by vaccination against Marek's disease and varied considerably between pairs of the sublines. Selection for resistance to subgroup A virus has been carried out in at least one commercial breeding company by intracranial inoculation of day-old chicks with RSV. The company claims that the selected meat-type line is approaching complete resistance to infection.

Second line of defence. The second line of defence, representing the ability of an infected bird to resist tumor development, is more complex than resistance to infection. Based on studies of regression of wing web tumors after inoculation with RSV, Gyles et al. (1968) proposed two mechanisms of this resistance. One mechanism influences resistance of cells to neoplastic transformation, while the other causes the initial tumor to regress. Later, 'regressor' lines of chickens were successfully selected (Gyles and Brown, 1971). Heritabilities of days-to-regression were estimated at .38 and of days-to-death after inoculation in a 'progressor' line at .26 (Gyles et al., 1981).

Collins and Zsigray (1984) summarized a large quantity of evidence for a clear association of some *B* haplotypes with regression of the Rous virus induced sarcoma (Table 33.1). The *Bu-1* locus influenced the rate of Rous sarcoma regression in some populations (Bacon et al., 1985).

Endogenous viral genes. Endogenous viral genes are believed to have arisen from germ-line integration of exogenous lymphoid leukosis viruses (Crittenden, 1981). More than 20 endogenous viral loci have been identified in White Leghorn chickens (Rovigatti and Astrin, 1983; Smith, 1986) and the latest edition of the *International Registry of Poultry Genetic Stocks* lists 26 of these genes (Somes, 1988).

Table 33.4 shows 22 of the endogenous viral genes, together with the length of the corresponding DNA fragments from digestions of chicken genomic DNA with two restriction enzymes. These fragments comprise part of the *ev* genes, together with the flanking sequence of the host genome. The phenotype associated with these genes ranges from no expression, through the expression of the envelope and core proteins, to the production of the complete endogenous virus. The chromosomal location is known for ten of the *ev* genes. It is believed that the variation in expression of the genes is a consequence of postintegration events in the evolution of the species. Although lymphoid neoplasms were observed in chickens free of infection with exogenous avian tumor viruses but harboring the endogenous virus, no evidence was found to suggest that the endogenous virus did directly induce the tumors (Crittenden et al., 1979b).

Initially, *ev* genes were found in all experimental and commercial populations of chickens tested (Rovigatti and Astrin, 1983), and it was speculated that the genes may play an essential developmental or physiological role in the host. However, finding of a single bird completely free of endogenous viral DNA sequences demonstrated that *ev* genes are not essential (Astrin et al., 1979a). The finding of four 'Italian partridge-colored' chickens free of such genes was reported by Gudkov et al. (1981). Gavora et al. (1989) reported that an entire inbred line of Leghorns, selected for Marek's disease resistance and high egg production, was free of *ev* genes. However, DNA sequences resembling the endogenous viral genome were recently found in chickens lacking *ev* genes (Dunwiddie et al., 1986), suggesting that under specific conditions of DNA hybridization, additional distantly related families of endogenous retroviruses may be identified. Sequences related to the avian retroviruses were also found in the genome of Japanese quail, a species previously considered free of endogenous avian leukosis virus elements (Chambers et al., 1986).

Antigenic similarity exists between the products of *ev* genes and exogenous lymphoid leukosis viruses. Therefore, chickens expressing *ev* genes may be immunologically tolerant to antigens common to exogenous and endogenous viruses (Crittenden et al., 1982, 1984, 1987). Harris et al. (1984b) reported reduced egg production and higher mortality in female progeny from dams carrying the sex-linked slow feathering *K* gene. It was postulated that the *K* gene may have facilitated infection with the lymphoid leukosis virus but no difference in immune competence in rapid and slow feathering chickens was found in a subsequent experiment (Bacon et al., 1986). The reason for the observation of Harris et al. (1984b) was later resolved by the finding that the *ev-21* and *K* genes are linked (Bacon et al., 1988). Alternatively, the *K* gene in White Leghorns may be a mutation resulting from the insertion of the *ev-21* in the feathering gene.

Table 33.4. Endogenous viral (*ev*) genes in chickens. (Modified from Smith, 1986).

ev gene number	Size of major SacI DNA fragment (Kb)	Size of 3' BamH1 DNA fragment (Kb)	Phenotype[1]	Reference	Chromosomal location
1	9.5	5.2	gs$^-$chf$^-$	Astrin (1978)	1
2	6.0	8.2	v-E$^+$	Astrin (1978)	2
3	6.3	7.3	gs$^+$chf$^+$	Astrin (1978)	Micro
4	8.7	7.3	gs$^-$chf$^-$	Astrin (1978)	1
5	19.0	13.0	gs$^-$chf$^-$	Astrin (1978)	1
6	21.0	4.4	gs$^-$chf$^+$	Astrin (1978)	1
7	13.0	7.6	gs$^-$chf$^-$	Astrin et al. (1979b)	2
8	18.0	23.0	gs$^-$chf$^-$	Astrin et al. (1979b)	1
9	23.0	11.0	gs$^-$chf$^+$	Astrin et al. (1979b)	
10	21.0	14.0	V-E$^+$	Astrin et al. (1979b)	
11	13.0	Not identified	V-E$^+$	Crittenden et al. (1983a)	
12	8.1	Not identified	V-E$^+$	Smith and Crittenden (1981)	
13	Not identified	Not identified	gs$^-$chf$^-$	Tereba (1981)	1
14	9.5	15.0	V-E$^+$	Hughes (1982)	
15	4.2	21.0	None	Wang et al. (1977)	
16	5.4	Not identified	None	Wang et al. (1977)	
17	11.0	Not identified	gs$^-$chf$^-$	Humphries et al. (1984)	
18	10.5	25.0	V-E$^+$	Humphries et al. (1984)	
19	7.6	9.8 or 18.0	V-E$^+$?	Humphries et al. (1984)	
20	8.1	9.8 or 18.0	V-E$^+$?	Humphries et al. (1984)	
21	9.2	20.0	Not identified	Humphries et al. (1984)	Z
22	17.0	21.0	Not identified	Smith and Crittenden (1986)	

[1] gs=group specific antigen, product of the *gag* gene; chf=chick helper factor, product of the *env* gene; V-E$^+$=complete endogenous virus; ?=not established with certainty.

The association of the *K* and *ev-21* genes may explain the difference in the incidence of cellular resistance to LL virus between the generally rapidly feathering White Leghorns, and meat chickens and brown egg layers that have significant frequencies of the slow feathering gene *K*. Ancestors of the meat and brown egg chickens, that carried the *K* and thus also the *ev* gene, would have been more susceptible to subgroups A and B of LL virus, and hence natural selection for resistance to infection would be more likely to occur (Cole, 1988, personal communication).

Leghorn strains selected for high egg production had a lower incidence of endogenous virus production than corresponding unselected control strains (Crittenden et al., 1979a). Two strains of Leghorns, free of exogenous LL viruses, had a similar average number of *ev* genes per bird, but the strain selected for resistance to Marek's disease and high egg production and egg weight carried five different *ev* genes as compared to seven such genes in the strain selected for MD susceptibility (Kuhnlein et al., 1988). Absent or reduced in the strain selected for resistance and production were the *ev* genes that code either for complete infectious endogenous virus or for the internal *gag* proteins. Thus selection for high egg production or Marek's disease resistance might have reduced the frequency of *ev* genes. Significant production trait differences were found between chickens with only silent *ev* genes, *ev* genes producing the group specific antigen, or *ev* genes producing the complete virus (Gavora et al., 1987). Consistent changes in the frequency of *ev* genes in strains selected for high egg production and related traits and for Marek's disease resistance were found by Kuhnlein et al. (1989), confirming that *ev* genes affect the host.

Other Retroviral Diseases And Interactions

Two lines of chickens previously selected for resistance and susceptibility to Marek's disease differed in response to tumorigenesis by the reticuloendotheliosis virus (REV). The MD resistant line tended to develop higher percentage of REV lesions than the susceptible line. Thus genetically determined resistance to REV may segregate independently of the major histocompatibility system (Scofield et al., 1978). Similar investigation in Japanese quail revealed the presence of significant genetic resistance to the reticuloendotheliosis virus (Terada et al., 1977).

When experimental and commercial stocks of chickens were compared for resistance to the avian myeloblastosis virus, involvement of both genetic and age resistance factors was indicated, and SPF chickens with no antibody against avian leukosis viruses exhibited the highest degree of the myeloblastosis virus proliferation (Heider et al., 1977).

Incidence of LL tumors and viremia was augmented in some White Leghorn lines given bivalent MD vaccines (Bacon et al., 1989). It was suggested that the vaccine herpes viruses may augment LL through their influence on bursal cells.

Oncogenes

Oncogenes were originally identified by genetic analyses of oncogenic retroviruses (Bister and Duesberg, 1982). They are not essential for virus replication. Most retroviruses with *onc* genes lack part or all of the three virion genes *gag*, *pol*, and *env* (Figure 33.3), and are replication-defective. However, in some, such as the RSV, an *onc* gene is a part of the genome of a replication-competent virus.

After the discovery of *onc* genes, their host cell counterparts were found in eukaryote genomes. The first such gene, *src*, was found in the avian genome by Stehelin et al. (1976). Oncogenes in the viral genomes are referred to as *v-onc* genes and their cellular equivalents as *c-onc* genes. The *onc* genes found in the genomes of domestic chickens are listed in Table 33.5. Additional *onc* genes have been identified in other species and will be likely also found in chickens.

Initially, the only known function of *onc* genes was to transform host cells neoplastically. Subsequently, products of *c-onc* genes have been identified as proteins with known physiological effects. The product of the *sis* oncogene was similar or identical to the platelet-derived growth factor (Waterfield et al., 1983). The product of the *c-erb* oncogene was found to be similar to the receptor for the epidermal growth factor (Downward et al., 1984). Thus oncogenes seem to play a normal physiological role and have importance in ontogenesis. The platelet-derived growth factor and the epidermal growth factor are known to be at high levels during *in vitro* changes that resemble neoplastically transforming cells.

Neoplastic transformation of cells involves alteration of oncogenes either in terms of their product or control of their expression. The *c-mos* in a nonvirally induced mouse myeloma was activated by a mechanism involving DNA transposition (Rechavi et al., 1982). In a human bladder carcinoma cell, a point mutation of a guanosine into thymidine activated an oncogene (Reddy et al., 1982).

Table 33.5. Oncogenes in the chicken genome. (Adapted from Somes, 1988).

Oncogene name	Viral equivalent
c-erb A	Avian erythroblastosis virus A
c-erb B	Avian erythroblastosis virus B
c-ets	E 26 leukemia virus
c-fps	Fujinami and PRC II sacroma virus
c-lil	A DNA sequence
c-mil/mht	Mill Hill 2 virus
c-myb	Avian myelocytomatosis virus
c-rel	Avian reticuloendotheliosis virus
c-ros	Avian sarcoma virus UR2
c-src	Rous sarcoma virus
c-ycs	Avian sarcoma virus Y73

Evidence that a *c-onc* gene can be activated by the insertion of a promoter was provided by Hayward et al. (1981). Their analysis of DNA and RNA from LL tumors showed that the LL provirus integrated adjacent to *c-myc*. The transcription originating from the viral promoter (long terminal repeat) enhanced the expression of *c-myc*, leading to neoplastic transformation. In a great majority of tumor cells the provirus was situated upstream from and in the same transcriptional direction as the *c-myc* gene (Fung et al., 1982). Integrations of the long terminal repeat of LL virus in bursal lymphomas corresponded to a region encompassed by two major hypersensitive sites of the normal *c-myc* gene (Schubach and Groudine, 1984).

Crittenden and Kung (1984) suggested that integration of avian leukosis viruses, that do not contain *v-myc*, near *c-myc* in bursal cells activates or promotes the gene's normal developmental switch-off through viral long terminal repeat. This interferes with the recombination of immunoglobulin heavy chain genes that normally causes a switch from the production of IgM to other Ig classes. Since both the avian leukosis and reticuloendotheliosis viruses can induce pathologically identical bursa lymphomas by *c-myc* activation, it is likely that the *onc* gene, and not the activation agent, controls the pathological manifestation of the activation (Crittenden and Kung, 1984). The finding that avian leukosis virus can also activate the *c-erb* gene by promoter-enhancing insertion, inducing a pathologically different neoplasm, supports this suggestion. Long terminal repeats do not seem to be essential for the tumorigenic process: *v-src* alone was shown to contain all the information necessary for sarcoma induction in chickens (Fung et al., 1983).

A comparison of the nucleotide sequence of the *c-myc* of chickens and humans reveals a high level of evolutionary conservation in terms of amino acid sequence (Watson et al., 1983). Such evolutionary stability also exists in other oncogenes underlining their physiological and developmental importance.

Newcastle Disease

Differences in resistance of Leghorn strains and sire families to Newcastle disease were demonstrated by Cole and Hutt (1961). Later Gordon et al. (1971) used chick mortality after challenge with the Newcastle disease virus as a criterion of a successful selection for susceptibility or resistance.

Salmonelloses

Two diseases of chickens, pullorum disease and typhoid, induced high mortality losses in poultry flocks from the 1920s to 1950s. It was shown that an early increase in body temperature of chicks by genetic selection reduced susceptibility to *Salmonella pullorum* (Hutt 1935; Hutt and Crawford, 1960). Comparison of resistance to challenge with *Salmonella pullorum* in 48-h-old chicks of Japanese native breeds and White Leghorns showed that White Leghorns were more resistant than the Onaga-dori or Hinai-dori breeds (Takayasu et al., 1966). White Leghorns were more resistant to *S. pullorum* than New Hampshire, White Plymouth Rock, or Japanese native breeds Hinai-dori or Onaga-dori (Hachinohe et al., 1966). Significant differences were also found among strains of Leghorns.

Breed differences in resistance of chickens to *Salmonella gallinarum* were observed by Lambert and Knox (1928) and Garren and Barber (1955). Prince and Garren (1966) found no differences in the bactericidal activities against *S. gallinarum* of whole blood or muscle and liver extracts from White Leghorn and Rhode Island Red chickens. The higher bactericidal activity of the intestinal extract from White Leghorn than from Rhode Island Red chickens was attributed to the higher activity of lysozyme. However, White Leghorn cockerels were more susceptible to a low virulence strain of *S. gallinarum* than Nigerian native birds. The native cockerels had a better response in hemoglobin, hematocrit, red blood cell count, and antibody titre (Aire and Ola Ojo, 1974). The authors suggested that the differences may have been due to an earlier development of the bursa of Fabricius in the Nigerian cockerels.

Although economically less important than *S. pullorum* and *S. gallinarum*, *S. typhimurium* can also produce high mortality in young chicks. Differences in resistance of inbred lines to *S. typhimurium* were reported by Bumstead and Barrow (1988). Crosses of a susceptible and a resistant line suggested the existence of an autosomal dominant resistance gene, and absence of maternal effects or influence of the MHC.

When a variety of lines of turkeys maintained free of *Mycoplasma*, *Salmonella*, and *Arizona* experienced an outbreak of fowl cholera, line differences in mortality were observed. The lines also experienced an outbreak of erysipelas with similar observations. Resistance was disease-specific: a line selected for high egg production was resistant to erysipelas and susceptible to fowl cholera. A line selected for increased body weight exhibited the highest mortality during both disease outbreaks. Thus different mechanisms may be involved in resistance to the two diseases (Saif et al., 1984).

Coccidiosis

Early evidence of genetic differences in resistance of chickens to coccidiosis was provided by Johnson (1927). Rosenberg (1941) found significant differences in survival of five breeds of chickens under coccidiosis challenge and Jeffers (1969) reported sex differences in resistance to *Eimeria tenella*. Several authors showed evidence that resistance to coccidiosis is, at least partly, associated with the MHC haplotype (Table 33.1). Mathis et al. (1984) compared resistance of chickens to *E. tenella* and *E. acervulina*. They observed significant variation among 18 sire families from a meat chicken randombred strain in body weight gain, packed cell volume (PCV), and coccidial lesion scores. Resistance to *E. tenella* was correlated with resistance to *E. acervulina*, and lesion scores were correlated with body weight gain and PCV. Hence lesion score may be a suitable selection criterion.

Leukocytozoonosis

Leukocytozoonosis is an epizootic disease caused by a protozoon *Leucocytozoon caulleryi*, transmitted by the biting midge *Culcoides arakawae*. It was first observed in Japan in 1954 and has since spread throughout the country. A two-way selection, under natural exposure, for antibody titre to *L. caulleryi* was carried out from Leghorn and Rhode Island Red base populations; it resulted in significant separation of the high and low lines in terms of antibody titre, with realized heritability of .15-.17 (Okada et al., 1988). Egg production in the summer, when the leukocytozoonosis epidemics occurred, was better in the low antibody than in the high antibody lines. This suggested that the high line is more susceptible to the infection, and the low average antibody titre in the low line reflects its resistance to the parasite.

Worms

In some countries, for example India, worms still cause losses to poultry populations. Breed differences in the period of maturation of worms and in total worm weight were found among chickens exposed to *Raillietina tetragona* infections. White Leghorns were generally more resistant than heavier breeds, particularly White Rocks (Nadakal et al., 1974). Resistance to *Ascaridia galli* was higher in New Hampshire than in White Leghorn, Cornish, and White Rock chickens (Buchwalder et al., 1977). Comparison of exposure at 30, 63, and 110 days indicated that the youngest age group had the most strongly pronounced clinical symptoms.

Mites

Mites cause considerable losses, mainly in egg producing chickens (De Vaney, 1978). Genetic variability in resistance to these ectoparasites was suggested by early observations that some hens have few or no mites in otherwise heavily infested populations (Payne, 1930). Males were more susceptible to mites than females (Abasa, 1965; Matthysse et al., 1974). A line of White Leghorns selected for high plasma corticosterone levels had lower mite infestations than a corresponding low corticosterone line (Hall and Gross, 1975). It was suggested that the sex difference can be in part attributed to higher levels of estrogen in females (Hall et al., 1978).

Eklund et al. (1980) scored the degree of infestation in both naturally exposed and artificially inoculated birds, and recorded their 40-week body weight and egg production. There were significant differences in average mite score between genetic groups. Males were more frequently and more severely infested than females. Body weight was positively correlated with mite score, whereas mite score and egg production appeared to vary independently.

Polygenically Inherited Developmental Disorders

Degenerative myopathy of the *Musculus supracoracoideus* (DMS). This condition, commonly called green muscle disease, pectoral myopathy, and hereditary muscular dystrophy, affects only the deep pectoral muscle which elevates the wing. The disease was first described in turkeys by Harper and Parker (1964), and in meat-type chickens by Harper et al. (1971). It affects mainly adult birds but has been also observed in seven-week-old broiler chickens (Richardson et al., 1980). Siller et al. (1979b) produced the disease experimentally by inducing birds to flap their wings until exhausted, thus confirming the noninfectious nature of the disorder. Fasciotomy of *M. supracoracoideus* prevented the development of the lesions, indicating that they are a consequence of a tight fascia that arrests circulation of blood when the muscle increases in volume due to exercise (Siller et al., 1979a).

Genetic differences in susceptibility to DMS in turkeys were first reported by Harper et al. (1975), and in chickens by Hollands et al. (1981). Faster growing birds with large breast angle are more susceptible to DMS than slower growing birds with less desirable body conformation (Grunder et al., 1984). This is in agreement with the results of Siller et al. (1979b) who induced DMS by forced wing exercise in turkeys and meat chickens but not in slower growing narrow-breasted egg chickens.

Leg weakness. This is a broad category of disorders causing important losses, particularly in broilers and turkeys. The causes of leg weakness can be infectious, such as encephalomyelitis virus, Marek's disease virus, viral arthritis, infectious synovitis, staphylococcosis, and also the contamination of Marek's disease vaccine with reticuloendotheliosis virus. Noninfectious causes include spondylolisthesis, tibial dyschondroplasia, and long bone distortions such as twisted leg, rotated tibia, chondrodystrophy and perosis (Riddell, 1980). Both genetic factors and environment have been implicated in long bone distortion problems (Haye and Simons, 1978).

Tibial dyschondroplasia (TD). This condition was more severe in chickens selected for high incidence of the defect than in those selected for low incidence, and most severe in birds fed a high chloride ration, thus indicating involvement of both genetic and environmental factors (Riddell, 1975). In a selection experiment, Sheridan et al. (1978) significantly increased the incidence of TD in broilers. Their results suggested involvement of maternal effects and presence of a recessive sex-linked gene associated with increased incidence of TD. TD was significantly lower in chickens heterozygous for the dwarfing gene (*Dw/dw*) than in their *Dw/Dw* counterparts (Lilburn et al., 1984).

Twisted leg. Genetic differences in the incidence of twisted leg, characterized by bending and twisting of tibia and metatarsus, were found in broilers (Haye and Simons, 1978; Hartmann and Flock, 1979). Leenstra et al. (1985) demonstrated that selection against twisted leg was effective in broilers reared in cages. The selection also reduced growth retardation and mortality due to twisted leg in broilers reared on litter.

Spondylolisthesis. This condition, commonly referred to as *kinky back*, causes posterior paralysis in commercial broiler chickens. Genetic predisposition to the disease has been suggested by Osbaldiston and Wise (1967), Riddell and Howell (1972), and Wise (1973). Riddell (1973) suggested that the condition is associated with high growth rate stimulated by commercial wheat-based broiler rations.

GENETIC IMPROVEMENT OF DISEASE RESISTANCE

Factors Influencing Improvement Of Genetic Aspects Of Health

Future improvement of genetic resistance to disease will be influenced by the state and structure of the poultry industry (Gavora, 1984). In most countries, genetic improvement is separated from reproduction and meat and egg production. Thus different health protection regimes can be used in each of the three segments of the industry. The flocks at the three levels could be controlled by the same organization, or each level may belong to a different concern.

The size of primary breeding stocks, numbering in the thousands, is small relative to the hundreds of millions of commercial hybrids produced from them. Therefore, breeding companies can introduce specific and expensive environmental conditions for their breeding stocks. They can reduce the protection of their breeding stocks by omitting vaccination, or they can deliberately expose the birds to disease agents. Alternatively, primary breeding stocks can be maintained in a specific-pathogen-free (SPF) environment. Such environment maximizes expression of genetic potential for production traits, but it reduces or eliminates information on resistance to disease. The fate of resistance would then depend on its genetic correlations with traits under selection (Gavora et al., 1974). Therefore, breeders should conduct occasional disease challenge tests and introduce selection for disease resistance into their programs as necessary. Eradication of congenitally transmitted pathogens in primary breeding stocks could result in their eradication from commercial hybrids. The poultry breeding industry seems to be moving toward maintenance of primary breeding stock under SPF conditions.

The world's major breeding companies in turkeys can be counted on the fingers of one hand, and in chickens the number is less than 20 world-wide. The trend is similar in waterfowl and guinea fowl. These narrowing gene pools may make the industry genetically vulnerable through reduced genetic variation in disease resistance traits. However, it is not clear how important such a reduction is, since genetic variation was not significantly reduced after more than thirty years of continuing multitrait selection (Gowe and Fairfull, 1985). Native breeds may be particularly important for future improvement of disease resistance. Such breeds may possess resistance genes unavailable in modern commercial hybrids. While the overall danger from the reduction of germplasm should not be overstated, poultry scientists, breeders, and producers need to be concerned. Maintenance of germplasm in poultry requires funding from private sources or government, which does not seem to be forthcoming. This is surprising, considering the well organized and funded efforts in plant breeding.

In most instances eggs or poultry meat are produced today in large production units. On the one hand, this concentration increases risks of contagious diseases and may also increase stress. On the other hand, modern housing, feeding, management, and disease control in such units compensate for the risks; health of intensively kept poultry is generally better than that in the production units of the past. The trend to further concentration and intensification of poultry production will likely continue, thus increasing the need for continued improvement of health protection. Simultaneously, because of animal welfare concerns, there is also a trend towards new 'deintensified' production conditions. Hence, requirements for genetic resistance to disease for the new type of facilities may change.

Genetic resistance is important in combination with other measures of disease control including sanitation, vaccination, medication, and eradication of infectious agents from poultry flocks (Gavora and Spencer, 1978; Gavora, 1984). Vaccination offers better protection in combination with genetic resistance, genetic resistance could be expected to improve effectiveness of medication, and genetic resistance is the only protection against drug-resistant pathogens. In addition, genetic resistance reduces vulnerability of populations from which a pathogen was eradicated, and protects when sanitation is inadequate.

Once accomplished, no further expenses are required for maintenance of genetic resistance, providing there is no substantial change in the virulence of pathogens or no unfavorable correlation of resistance with trait(s) under selection. As well, substitution of genetic resistance for vaccination and use of drugs may become important in light of increasing consumer concerns.

Genetic Parameters Of Resistance To Disease

Estimates of heritability of total mortality or viability, and resistance to specific diseases are summarized in Table 33.6. Wherever possible, the estimates are based on sire components of variance. Where authors reported both binomial and normal scale heritabilities, the normal scale estimates are shown. Estimates of heritability are expected to be identical regardless of whether the trait is expressed as mortality or viability.

Heritability of total mortality (deaths from all causes) is generally lower than that of resistance to specific diseases (Table 33.6). The average heritability of total mortality in the table is .07 while the average of heritability of resistance to specific diseases is .25. Total mortality includes all causes of death, from drowning and cannibalism to death from infectious diseases. Therefore, it must also encompass a great variety of disease resistance mechanisms. It is not surprising that the heritability of this poorly defined trait is low. A narrower range of resistance mechanisms is involved in any one of the specific diseases, and consequently the heritability of resistance is higher.

In contrast to heritability estimates, the definition of disease traits is critical for estimates of genetic correlations. A correlation with resistance would have the opposite sign to a correlation with susceptibility to the same disease. There are three types of genetic correlations involving disease resistance: between resistance to two different diseases, between resistance to a disease and a production trait, and relationships of genetic markers to disease resistance.

Attempts to study relationships between resistance to various diseases date back to 1951, when Carson (1951) tested the resistance of lines selected for resistance and susceptibility to 'leucosis' (mostly Marek's disease) to a number of pathogens. He concluded that resistance or susceptibility to 'leucosis' was not indicative of resistance to *Pasteurella avicida, Eimeria tenella,* or *Salmonella pullorum.* Similarly, Patterson et al. (1961) compared inbred lines of White Leghorns selected for resistance or susceptibility to cecal coccidiosis or 'visceral lymphomatosis' (mostly lymphoid leukosis). The lines were challenged with *Eimeria tenella, E. necatrix, E. maxima, E. brunetti, Ascaridia galli,* and *Raillietina.* In addition, sufficient chronic respiratory disease occurred in the flock to permit comparison of resistance. The authors concluded that selection for resistance to one disease did not necessarily result in resistance to the other diseases tested.

A preliminary evaluation of data from trials in which multiple genetic groups of chickens were exposed to a number of pathogens and to stress did not indicate strong genetic correlations between resistance to the various agents (Gavora and Spencer, 1983). Nevertheless, the study indicated compatibility of resistance to various diseases, since

Table 33.6. Heritability of total mortality, viability, and resistance to specific diseases in chickens.

Trait		Heritability estimates	References
Total mortality (viability)			
Embryonic		.01	Wilson (1948)
Brooding	(0-6 wks)	.01 to .03	Morris (1959)
Rearing	(11-20 wks)	.02	Dickerson (1957)
Adult	(21-60 wks)	.05	Dickerson (1957)
	(61-70 wks)	.00	Dickerson (1957)
	(annual)	.08	Lush et al. (1948)
	(annual)	.08	Robertson and Lerner (1949)
	(laying house)	.08	Hale (1959)
	(laying house)	.06	Hill et al. (1954)
	(annual)	.03	Hays (1954)
	(141-273d)	.00 to .38	Gavora et al. (1974)
	(141-497d)	.00 to .27	Gavora et al. (1974)
Resistance to specific diseases			
Marek's disease		.67	Friars et al. (1972)
		.14	Von Krosigk et al. (1972)
		.61	Gavora et al. (1974)
		.06 to .18	Hartmann and Sanz (1974)
Newcastle disease		.07 to .17	Gordon et al. (1971)
Mites		.06 to .26	Eklund et al. (1980)

some genotypes were resistant to more than one agent. Hence, simultaneous improvement of resistance to various diseases should be possible.

As mentioned earlier, resistance to reticuloendotheliosis seemed to be unrelated to resistance against Marek's disease (Scofield et al., 1978). Strains of chickens relatively resistant and susceptible to Marek's disease differed in ability to regress Rous sarcoma tumors, but the differences were not consistent with their genetic resistance to Marek's disease (Calnek et al., 1975). On the other hand, there was a high genetic correlation between resistance of chickens to a medium and a highly virulent isolant of Marek's disease virus (Gavora et al., 1984).

From a practical point of view, it is not the resistance to individual diseases but the ability of a bird to withstand challenge by multiple pathogens in a practical production environment that determines commercial profitability. It depends on a multiplicity of environmental, genetic, and epigenetic factors. Natural exposure to disease agents is particularly important as a test of resistance where diseases specific to the local environment exist. For example, Boa-Amponsem et al. (1985) examined mortality data from four international commercial egg production hybrids and one local commercial bird. Overall, the locally developed genetic group was the least susceptible to most of the diseases that were typical for Ghana.

Most genetic correlations shown in Table 33.7 were estimated from studies in which both the mortality or disease resistance and production traits were measured in the same population. Results from these studies have to be interpreted with caution because of possible 'built-in' relationships. Birds affected by a disease will have a reduced production performance regardless of their genetic potential for the production trait measured. Only in one study was egg production measured in a flock of females vaccinated for Marek's disease, while resistance was measured in their sisters challenged with a Marek's disease virus (Gavora et al., 1974).

It is not surprising that a high negative correlation was found between adult mortality and hen-housed egg production, which is affected by mortality (Hill et al., 1954). The genetic nature of such a relationship is confirmed by the correlations of adult mortality or adult viability with hen-day percent egg production, a trait that is less influenced by mortality (Table 33.7). Naturally, the opposite signs of the correlations between mortality and production and viability and production indicate the same overall tendencies.

Resistance to Marek's disease appears to be genetically correlated with early sexual maturity, high egg production, and low egg and body weights, or body weight gain. The genetic correlations of Marek's disease resistance with production traits suggest to the breeder that selection in egg stocks that usually emphasizes high egg production and low body weight should not be detrimental to MD resistance. However, strong selection emphasis on increased egg weight or body weight may lead to reduced resistance to Marek's disease. No consistent genetic correlations were found between egg quality traits and MD resistance (Gavora et al., 1974).

An interesting connection between activity, measured by an open-field test, and resistance to coccidiosis was suggested by Faure and Yvore (1980). An experimental infection with *Eimeria tenella* affected more severely the active than the inactive strain of chickens.

Because of cost and humane considerations, it is desirable to replace direct selection for

Table 33.7. Genetic correlations of mortality or disease resistance with production traits in chickens.

Disease resistance trait		Production trait	Genetic correlation	Reference
Adult mortality	-	Hen-housed egg production	-.56	Hill et al. (1954)
	-	Hen-day percent egg production	-.27	Hill et al. (1954)
Adult viability	-	Hen-day percent egg production	.42	Nordskog and Hill (1958)
Marek's disease mortality	-	Hen-day percent egg production	-.18	Von Krosigk et al. (1972)
	-	Age at sexual maturity	.15	Von Krosigk et al. (1972)
	-	8-week broiler weight	.26	Hartmann and Sanz (1974)
Marek's disease resistance	-	Hen-housed egg production	.23 to .41	Gavora et al. (1974)
	-	Hen-day percent egg production	.31 to .36	Gavora et al. (1974)
	-	Egg weight	-.24 to -.53	Gavora et al. (1974)
	-	Age at sexual maturity	-.08 to -.46	Gavora et al. (1974)
	-	Adult body weight	-.37 to -.43	Gavora et al. (1974)

resistance to disease based on challenge tests by indirect selection based on suitable markers. Unfortunately, with the notable exception of the MHC as a marker for resistance to Marek's disease, other attempts to find suitable markers have been unsuccessful. No relationship was found between the dwarfing gene and susceptibility to *E. coli* (Mauldin et al., 1981). Studies of relationships between susceptibility of chicks to *E. coli*, *Salmonella pullorum* or *Mycoplasma gallisepticum*, and the *A*, *B*, *C*, *D* and *E* blood group systems, an arbitrarily designed locus *x5*, and the *E* locus for feather color, revealed differences in resistance associated with some alleles in individual breeds, but no universal relationships across the populations were observed (Gintov et al., 1978). Miller et al. (1972) attempted to find correlations between mortality of chickens after injections of gold salts and their resistance to MD and LL. The relationships were inconsistent: positive correlations in some strains, none in others.

Pavel (1975) and Pavel et al. (1979) attempted to develop an index for nonspecific resistance. Components of the index included bactericidal activity of serum for *E. coli* and *Staphylococcus aureus*, lysozyme activity of serum for *Micrococcus lysodeiticus*, as well as rate of skin inflammation, protein content in serum, and content of hemoglobin in blood. It was concluded that no universal characteristics of general resistance were identified (Pavel et al., 1985).

Changes of gene frequencies at five polymorphic loci were examined in lines of chickens selected for MD resistance over five generations. The frequencies of blood group and MHC alleles A^6 and B^8 showed a consistent increase and B^{11} a gradual decrease. An allele of alkaline phosphatase increased significantly in two of the seven selected lines (Okada et al., 1977).

Effects Of Pathogens On Heritability Of Production Traits And On Selection Gains

Disease can affect heritability and cause reduced genetic progress from selection (Gavora et al., 1983). The heritability of production traits in populations affected by disease may be reduced because of an increase in phenotypic variance due to disease. The increase $E(\Delta_V)$ can be estimated as:

$$E(\Delta_V) = d^2p(1-p) + pVd,$$

where d is the change in performance caused by the disease in a proportion p of the population, and Vd is the variance of such change.

Congenital transmission of the pathogen or genetic correlation of resistance and production traits could result in an increased variance component for the parent contributing to the transmission, with consequent effects on estimation of heritability of production traits from sire or dam variance components. Gavora et al. (1983) provide examples of these situations in flocks of hens infected with LL virus.

Congenital infection of birds by an agent with negative effects on the trait(s) under selection may result in 'false' genetic gains generated as follows (Gavora et al., 1980). Lower performance of individuals affected by the disease results in their elimination in the process of selection. This reduces the frequency of infected individuals in subsequent generations and causes a corresponding increase in the mean population performance compared to the base or unselected control population, a false genetic gain. As soon as selection is relaxed, and providing that the disease agent has not been completely eradicated, the false genetic gain is reduced or eliminated. In commercial breeding, false genetic gains may be generated in the primary breeding stocks. The gains are lost in the process of producing the commercial hybrid under relaxed selection in grandparent and parent stocks. Although the size of the false gains is usually not large, the commercial hybrid may show a disappointing performance compared to the original primary breeding lines.

A computer simulation of these processes suggested that individual selection is more efficient than primary selection in eliminating the affected individuals and that size of the false genetic gains is more significant with regard to short-term than to long-term selection (Harris et al., 1984a). This is because the size of the false genetic gains remains relatively constant and their importance, relative to total genetic gain, diminishes with the increase of cumulative genetic gains under long-term selection.

Breeding For Disease Resistance

The methods of selection for disease resistance are summarized in Table 33.8, taking into consideration the necessity of exposure to pathogens for any direct selection. The simplest and widely used approach is the observation of breeding stocks and its use in selection. This approach does not introduce negative effects on the expression of genetic potential for production, but it does not guarantee adequate expression of genetic resistance. As a rule, the breeder will try to minimize the exposure of his breeding stock to disease. Hence, paradoxically, resistance to important diseases may not be expressed. In the absence of adequate diagnostic capabilities, breeders tend to select against total mortality, a trait that is poorly defined and that has low heritability. Nevertheless, improvement of viability can be achieved by this method, particularly if irrelevant mortality (such as accidents and trauma) is eliminated and family mean mortality data are used in selection among survivors.

The second method in Table 33.8 requires a challenge of breeding stocks with infectious agents. This may have a negative effect on the expression of genetic potential for production traits and also represent a danger of losing, due to disease, breeders with high genetic potential for production traits. Nevertheless, the method is occasionally used. For example some breeders brood young chickens on litter from hens to get them exposed to coccidia and then select on coccidiosis resistance. This method could result in good expression of disease resistance, particularly if dosage of the pathogen can be standardized.

Exposure of sibs or progeny of the breeding stock to the disease agent (Table 33.8) is an excellent way to measure disease resistance. The challenge dose can be standardized and administered at an optimum level. The challenged populations are not used for production or breeding, and high losses may be acceptable. This approach had been used by poultry breeders to improve MD resistance prior to development of vaccines. Its obvious disadvantage is the high cost of challenged populations, as well as the need for isolation facilities for challenge tests. In addition, deliberate challenge with pathogens may be objectionable from the animal welfare point of view.

Dickerson (1968), assuming largely additive gene effects, no environmental relations among sibs, and equal total numbers exposed, found the response expected per generation from optimum early progeny challenge test selection to be essentially equal to that expected from full-sib test selection in experimental situations. However, he noted that in commercial breeding programs, selection based on exposure testing of full-sibs would have higher expected response, and would allow reduced intervals between generations by about one fourth, compared with early progeny test selection.

Indirect selection based on suitable genetic markers (Table 33.8), represents an ideal approach to the improvement of disease resistance since it does not require exposure to

Table 33.8. Methods of selection for disease resistance. (Modified from Gavora and Spencer, 1983).

Type of selection	Method	Effects on production traits	Expression of resistance	Cost
Direct	Observe breeding stock	None	Questionable	None
	Challenge breeding stock	Negative	Good	Low
	Challenge sibs or progeny of breeding stock	None	Good	High
Indirect	Use markers of disease resistance	None		Low

pathogens. The effectiveness of indirect selection, relative to selection based on challenge with a pathogen, depends on the heritabilities of the marker and resistance traits and their genetic correlation. As a general rule, it is desirable to search for highly heritable marker traits that are strongly correlated genetically with resistance to disease (Gavora and Spencer, 1983).

The first successful selection for resistance to disease was reported by Lambert (1932), who selected White Leghorns for resistance to fowl typhoid by standardized challenge of chicks with *Salmonella gallinarum*. Years later, the resistance-selected population was retested, and its resistance was found to be unchanged despite many generations without challenge or selection (Cole, 1988, personal communication). This confirms persistence of genetic resistance. Bearse et al. (1939) reported on eight years of selection for disease resistance and susceptibility in White Leghorns, and 15 years of selection for viability in White Leghorns was described by Moultrie et al. (1953).

The report by Hutt and Cole (1947) on successful selection for resistance and susceptibility to avian lymphomatosis marked the start of a number of similar selection studies over the next 40 years. When Hutt initiated the selection experiment in 1935, avian lymphomatosis included both Marek's disease and lymphoid leukosis, and only much later were differential diagnostics for the two diseases developed. Nevertheless, it appears that the primary selection by Hutt and Cole was on resistance to Marek's disease. In the 1935 base population, 14.6 percent of the White Leghorn birds died of neoplasms. In the tenth selected generation, deaths from neoplasms in the resistant strain K were 7.9 percent and in the resistant strain C they were 8.3 percent; strain S, selected for susceptibility, reached 34.9 percent mortality. In 1951, females of the C and K resistant strains died only at 2.7 percent, while 61 percent of the susceptible strain S died with lymphoid tumors (Hutt and Cole, 1953). A global evaluation of these selection experiments spanning more than 30 years (Cole and Hutt, 1973) provided evidence that multitrait selection in the two closed strains C and K raised viability, egg production, and egg size, and established a high degree of resistance to Marek's disease. Strain S, selected for MD susceptibility, developed a uniform and high level of MD susceptibility, and its losses under field exposure to MD virus ranged from 50-60 percent, while up to 100 percent of the birds died when challenged with virulent laboratory strains of the virus.

Cole (1968) used response of a sample of progeny to inoculation with MD virus as the criterion for selection of breeders, and developed a resistant line N and a susceptible line P with respective susceptibilities of 12.9 and 90.7 percent after only two generations of selection from a common stock in which the level of susceptibility was 51.1 percent. In the fifth selected generation, the susceptibilities reached 5.0 and 97.7 percent in lines N and P respectively (Cole, 1972). MD susceptibility of line N ranged from 7-15 percent and that of line P from 53-94 percent after challenge with four different isolants of MD virus (Cole, 1969). Selection for resistance to artificial challenge with MD virus reduced the incidence of MD by 14 percent in the selected line, as compared to the control line of a commercial female broiler strain of chickens (Friars et al., 1972).

Simultaneous improvement of commercially important traits of egg chickens and resistance to MD was studied by Gavora and Spencer (1983). The selection for egg production and viability resulted in improved MD resistance, as well as production gains. Direct selection for MD resistance, in addition to the production traits, further dramatically improved survival. A combination of selection for genetic resistance with vaccination resulted in the best protection against MD. Egg production potential was expressed better in vaccinated than nonvaccinated stocks. Without vaccination, the resistance-selected strains significantly outperformed the production-selected strains because of their improved MD resistance. Strain crossing resulted in additional gains in both egg production and MD resistance.

Successful selection for resistance or susceptibility to Newcastle disease virus (NDV) was reported by Gordon et al. (1971). However, realized heritability estimates for resistance to NDV were low (Table 33.6).

Selection in a strain of White Leghorns for resistance and susceptibility to acute cecal coccidiosis caused by *Eimeria tenella* resulted in a six-fold difference in mortality rates of the two lines (Johnson and Edgar, 1982). Relaxation of selection for resistance resulted in gradual regression of the mortality rates toward that of the base population. Large doses of oocysts resulted in spreading of the infection in the resistant line beyond the typical site. The chief foci of infection remained unchanged in the susceptible line. Thus selection for resistance may have disrupted the typical host-tissue specificity of the parasite.

Molecular Genetic Approaches To The Improvement Of Disease Resistance

Information on the avian genome at the molecular level is rapidly increasing. As of 1987, there were 110 avian genes or gene families that had been cloned and there were 356 avian gene-sequence entries in the GENBANK (Gavora and Seligy, 1988).

The first successful gene transfers in the chicken have been accomplished. Injections of reticuloendotheliosis virus, or recombinant avian leukosis viruses, containing the subgroup A *env* coding region, into the blastoderm of unincubated fertilized chicken embryos resulted in successful insertion of the retroviral genetic information into the germ line of chickens (Salter et al., 1986, 1987). The transgenic chickens so obtained were viremic for the recombinant virus. Later, individuals with a proviral insert that was defective for and incapable of production of a complete virus were detected (Salter and Crittenden, 1987). Such chickens expressed the subgroup A viral envelope glycoprotein and were resistant to subgroup A viruses (Crittenden, 1988, personal communication). This significant achievement represents an example of a potential commercially applicable gene transfer.

The recessive resistance to infection with avian leukosis viruses occurring naturally is due to lack of suitable receptors on the host cell surface, as discussed above. In contrast, these transgenic chickens carry a dominant resistance, since a single copy of the transgene is sufficient to produce the envelope glycoprotein that blocks the cell receptors. This type of resistance is more desirable because it may allow the poultry breeder to use a homozygote resistant line in the production of the final heterozygote resistant commercial hybrid.

New routes for the application of DNA manipulation in the improvement of resistance to disease are emerging from the report on anti-sense RNA (Izant and Weintraub, 1985). They demonstrated that putting a cloned gene in a backwards orientation to its promoter results in transcription of anti-sense RNA that can then inactivate the normal messenger RNA, preventing gene expression. Transfer of genes for anti-sense RNA may have potential in blocking specific steps in viral infections.

The use of restriction fragment length polymorphisms (RFLP) to identify the individual endogenous proviral genes of the chicken, represents an example of how RFLP could be utilized in future poultry improvement. If *ev* genes are conclusively proven to be undesirable, breeders can select against their presence on the basis of the DNA test. Similarly, RFLP should be applicable in other areas, in particular in the determination of major histocompatibility haplotypes, where they will provide a tool for selection under both experimental and commercial conditions.

Meaningful contributions of molecular genetics to poultry improvement will only come from the integration of the new methods with conventional approaches. Combined molecular genetics expertise and expertise in poultry breeding and disease resistance, as well as facilities for measuring both quantitative production traits and disease resistance in sufficiently large populations, are essential prerequisites for identification of the problems or traits to be subjected to biotechnology research, production and extensive testing of transgenic birds, and integration of transgenics and new procedures involving DNA analysis and manipulation into conventional breeding programs to maximize overall genetic improvement (Crittenden and Gavora, 1986).

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Chapter 34. NUTRITIONAL GENETICS

R.A.E. Pym

All farm livestock species require essentially the same dietary nutrients although there is some variation in the amounts of specific nutrients required to promote optimal growth, reproductive performance, milk production, wool growth, etc. Variation in nutrient utilization within a species is similarly restricted to quantitative rather than qualitative differences, with the exception of certain metabolic disorders where the affected individual may be unable to utilize a nutrient or metabolite.

There are two related yet distinct components of nutrient utilization in poultry. These are *nutrient requirements* and *food utilization efficiency*. The nexus between the two is demonstrated by the fact that the efficiency with which the bird converts food into either body tissue or eggs depends upon the specific nutrient requirements of the bird and on the supply of those nutrients in the food. *Nutrient requirements* are generally determined as the quantity of a specific nutrient per unit weight of food, per unit Metabolizable Energy (ME) concentration in the food, or per day, necessary for optimal growth or egg production. *Food utilization efficiency*, by way of contrast, is a composite measure of the efficiency with which all the nutrients supplied in the food are utilized for the specified form of production; it is markedly affected by dietary energy concentration. The relative food conversion efficiency of individual birds, or different strains of birds with different specific nutrient requirements, may thus be affected by the nutrient composition of the diet used. In this chapter, nutrient requirements and food utilization efficiency will be considered under separate headings.

Reports of genetic variation in nutrient utilization in poultry are numerous and extend back in the scientific literature for over 40 years. There have been a number of reviews of the component fields of this general area of study. Genetic aspects of nutrient requirements in poultry have been reviewed by several authors (Hutt, 1961a, 1961b; Olsen et al., 1964; Nesheim, 1966, 1975; Quisenberry, 1969; Morris, 1972; Hvidsten and Kohlstad, 1972; Sorensen, 1985) and genetics of efficiency of food conversion for growth and/or egg production have been more recently reviewed by Fairfull and Chambers (1984), Pirchner (1985), Pym (1984, 1985) and Luiting (1987). The very excellent review by Nesheim (1975) of genetic variance in nutritional requirements in poultry covered published information on that topic in considerable breadth and depth, although since then there have been a number of studies which have provided additional information on the genetic control of requirements for specific nutrients.

ENERGY UTILIZATION

In the classical sense, it is difficult to define energy requirements in terms of dietary concentration for either growth or egg production, since the bird has a remarkable propensity for regulating its energy intake across a wide range of dietary energy concentrations (Morris, 1968; Fisher and Wilson, 1974). In this regard energy is a unique nutrient. The influence of dietary energy upon food utilization efficiency, however, is profound because of the bird's ability to regulate energy intake and the fact that liveweight gain is to a large extent proportional to energy intake. As such, it is appropriate to consider energy utilization under the heading of food utilization efficiency.

FOOD UTILIZATION EFFICIENCY

Since food accounts for about 70 percent of production costs, the efficiency with which birds convert food into body tissue or eggs is a major determinant of profitability of both broiler and egg production. Notwithstanding this, commercial breeders have until recently relied almost exclusively on correlated response to selection for gain in broilers, and for egg mass production in layers, to achieve genetic improvement in food utilization efficiency. There is now considerable interest in direct selection for food utilization efficiency in both industries. The main problems associated with the use of this trait in commercial selection programs are the many ways in which efficiency can be expressed and measured, and the limited information available as to the consequences of alternative selection strategies.

Food utilization efficiency, or feed efficiency as it is frequently termed, is a measure of the efficiency with which food is converted into body tissue or eggs and is normally expressed as the weight ratio of food consumed per unit of liveweight gain or egg production (or per dozen eggs). This measure is generally referred to as the *feed conversion ratio* or FCR. It is in some ways unfortunate that this measure should have been chosen as the normal means of expressing feed efficiency in poultry since, because of the arrangement of the traits in the ratio, it is actually an inverse measure of food conversion efficiency. Moreover, this and the inverse expression take no account of the energy content of the food or of the product and are thus not measures of *energetic efficiency*, which Brody (1945) defined as "the ratio of the desired form of output energy to the given form of input energy." Further, FCR is a measure of *gross* rather than *net* efficiency since the food consumption component of the ratio is utilized for purposes of both maintenance and production. This concern has prompted some workers to make correction for the maintenance component; for instance Koch et al. (1963) advocated abandoning the ratio technique of determining feed efficiency for growth in livestock on the grounds that the ratio is a biased estimate. They suggested various midweight adjustments for gain and food consumption.

It would be appropriate to consider food utilization efficiency for growth and egg production under separate headings since there are large differences in the components of production and in the ages and types of birds and their nutrient requirements.

FOOD UTILIZATION EFFICIENCY FOR GROWTH

Measurement Of Feed Efficiency

Food consumption per unit gain and its inverse have been shown to have different coefficients of variation and heritabilities in studies with pigs (Robison and Berruecos, 1973) and mice (Timon and Eisen, 1970), and there has been some debate as to which is the preferred measure.

Measurement interval can have a considerable influence on the relative food conversion efficiency of individuals. Weight-constant measures of efficiency favor the faster growing birds since the growth period is shorter and maintenance requirements commensurately reduced. These birds are, however, penalized with age-to-age measures since they carry a heavier average weight, with its greater maintenance cost, for the time-constant period.

Since birds are slaughtered at given weights it is appropriate that measures of efficiency be made at those weights. With commercial selection for food utilization efficiency, however, because of the large number of individuals under test, it would be appropriate to test the birds over a time-constant period and make adjustment for mid- or final liveweight such that the outcome of selection would be to optimize efficiency to that final weight (Chambers and Lin, 1988).

Inheritance Of Feed Efficiency

Hess et al. (1941) produced the first published evidence on the inheritance of feed efficiency in which the same sires were shown to produce the most efficient purebreds and crossbreds. They and others (Glazener and Jull, 1946; Hess and Jull, 1948: McCartney and Jull, 1948) used the formula $E = C - kW$ in studies of the inheritance of feed efficiency and its relationship to food consumption and body weight in chickens. The formula is a derivative of the equation for the curve of 'diminishing increment'. In the equation, E was gain/food consumption, W was $(W2-W1)/2$ where W1 and W2 were the liveweights at the beginning and end respectively of the biweekly period, and C and k were constants whose values were solved by least squares analysis. C was the theoretical maximum efficiency if no food was used for maintenance, and k was the maintenance requirement per unit of weight. Both Hess and Jull (1948) and McCartney and Jull (1948) found differences in C and k values between the progeny of different sires, which provided evidence for the inheritance of feed efficiency independent of liveweight, rate of gain, or time.

Fox and Bohren (1954) proposed an alternative regression of mean efficiency on mean body weight (which has a positive slope) for studying between-bird or between-breed differences in efficiency. They noted a significantly larger coefficient for the Leghorn strain tested than for three heavy breeds.

Since these earlier investigations, and since the development and refinement of quantitative genetic theory, there have been a number of studies of the inheritance of food utilization efficiency. Sib analysis and parent-offspring regression estimates of the heritability of FCR measured over varying growth intervals have ranged from about 0.20 to 0.80 (Thomas et al., 1958a; Wilson, 1969; Pym and Nicholls, 1979; Chambers et al., 1984; Leenstra et al., 1986; Leenstra and Pit, 1988), with a mean of about 0.4 to 0.5. Realized estimates for the heritability of time-constant measures of feed efficiency have generally been somewhat lower than the above statistical estimates, ranging from 0.18 to 0.56 with a mean of about 0.25 (Guill and Washburn, 1974; Pym and Nicholls, 1979; Pym, 1985; Chambers, 1987).

The relationship between feed efficiency and liveweight gain is of both biological and economic significance, since until recently genetic improvement in food utilization efficiency in commercial broiler chickens has been achieved almost entirely as a response to the shorter growth period achieved through selection for increased growth rate.

Correlation Between Liveweight Gain And Food Utilization Efficiency

Faster growing birds either within or between strains are more efficient in converting food into body weight than their slower growing counterparts (Glazener and Jull, 1946; Hess and Jull, 1948; McCartney and Jull, 1948; Fox and Bohren, 1954). Published estimates of the phenotypic correlation between weight gain and FCR have ranged from about -0.4 to -0.7 (Wilson, 1969; Guill and Washburn, 1974; Pym and Nicholls, 1979; Leenstra et al., 1986), and Chambers et al. (1984) obtained an estimate of +0.52 for the correlation between weight gain and gain/food consumption. Sib analysis or parent-offspring regression estimates of the genetic correlation between weight gain and FCR measured over various intervals have ranged from about -0.25 to -0.70 (Wilson, 1969; Pym and Nicholls, 1979; Leenstra et al., 1986), and Chambers et al. (1984) obtained an estimate of +0.69 for the genetic correlation between gain and gain/food consumption. The difference in sign between the two sets of estimates is due entirely to the arrangement of traits in the feed efficiency ratio and not to any biological difference. Thus, while faster growing birds convert food into body weight more efficiently than their slower growing counterparts, differences in growth rate account for no more than half of the variation in feed efficiency measured over a time-constant interval.

Physiological Components Of Food Utilization Efficiency

Food utilization efficiency is a complex trait influenced by a number of component physiological traits which contribute to either direct or correlated response in food utilization efficiency.

Improvement in gross efficiency can presumably be achieved by one or more of the following effects:

1. A reduction in external losses. This includes spillage as affected by feeding behavior which has a genetic component (Siegel et al., 1984a). This effect can be reduced by the use of well designed and managed feeders.
2. An increase in food intake allowing proportionately more food to be used for growth purposes after maintenance requirements have been met. There is no improvement in the net efficiency of food utilization for growth, and it is likely that this mechanism is responsible for much of the correlated improvement in gross efficiency to selection for increased growth rate.
3. An increase in the digestibility or metabolizability of dietary nutrients. It has been a general assumption that there is little variation in digestibility or metabolizability of dietary nutrients within or between different strains or breeds (Fowler, 1962; Blaxter, 1968; Sutherland et al., 1974). There have been, however, a number of studies with chickens which have demonstrated differences in metabolizability of dietary energy between strains and breeds (Sibbald and Slinger, 1963), and between lines of chickens selected for divergent 8-week liveweight (Proudman et al., 1970) or for feed efficiency and its components (Pym et al., 1984).
4. A reduction in maintenance energy requirements. Selection for both increased liveweight gain and improved food utilization efficiency in chickens has been shown to result in lowered maintenance requirements, while selection for increased appetite has had the opposite effect (Pym and Farrell, 1977; Pym et al., 1984). Factors contributing to variation in maintenance requirement include activity, feathering and hence insulation to heat loss, and body composition.
5. An increase in the availability of metabolizable energy (ME) for gain. One factor contributing to this effect is the differential cost of fat and protein deposition. Since fat is deposited with a much greater energetic efficiency than protein (89 compared to 65 percent according to Pym and Farrell, 1977), it is *energetically* more efficient to deposit fat than protein. However, because of the higher energetic density of fat compared to protein (39 compared to 23 MJ/kg), on a dry weight basis the two are deposited with

much the same energy cost.

6. A reduction in the energy content of the gain, that is, partitioning of water, fat and protein in the body. Because protein in muscle contains about 70 percent water while fatty tissue contains only about ten percent, and since the two are deposited with roughly the same energy cost on a dry weight basis, it should in theory cost about three times as much energetically to deposit the same weight of fatty tissue as muscle. Selection for improved food utilization efficiency has been shown to reduce the proportion of body fat (Washburn et al., 1975b; Pym and Solvyns, 1979), while selection for reduced body fat has been shown to result in an improvement in food utilization efficiency (Leclercq et al., 1980; Whitehead and Griffin, 1984).
7. A reduction in protein breakdown rate. Protein turnover rate is high, which gives rise to the low energetic efficiency of protein deposition compared to fat. Selection for improved food utilization efficiency has been shown to reduce protein breakdown rate, while selection for increased appetite had the opposite effect (Tomas et al., 1988).

Selection For Food Utilization Efficiency For Growth

Before 1980 there were only three published studies of selection for food utilization efficiency in chickens (Wilson, 1969; Guill and Washburn, 1974; Pym and Nicholls, 1979). Since then, the results of three additional studies have been published (Sorensen, 1984; Leenstra and Pit, 1987; Chambers, 1987), providing a reasonable body of data and information for commercial meat chicken breeders to make decisions relating to the most appropriate methods to use and the likely consequences of such selection. Such prediction is unquestionably made difficult because of the complexity of the trait. One particular benefit of the more recent studies is that in each case the selected lines have been derived from either present-day commercial stocks or birds with similar rapid growth rates. One valid concern about the earlier studies is that the birds used were very much slower growing than today's broiler chickens, and that direct and correlated response in present-day commercial nucleus lines may be quite different. Most commercial breeders now include some measure of direct selection for food utilization efficiency in their broiler breeding programs.

The above six studies included a number of different comparisons of selection for food utilization efficiency, in all cases measured over a time-constant interval, either in single-trait or multiple-trait selection lines.

Direct and correlated response in the performance traits. Wilson (1969) obtained an average realized genetic correlation of -0.51 between weight gain and FCR from one generation of selection in two lines of chickens for either 5-week to 10-week weight gain or decreased FCR measured over the same interval. He estimated selection for gain to reduce the amount of food needed to produce one unit of gain to be about 75 percent as much as direct selection for feed efficiency.

Guill and Washburn (1974) selected three broiler lines and one randombred population for either high or low FCR, each with body weight either unrestrained or held constant. In the lines selected for low FCR, there was virtually no difference in response between the body weight-constant and body weight-varied lines in both populations. The realized heritability was similar within the same class of stock for the body weight-constant and varied lines, whereas the overall heritability of FCR was higher (0.42) in the randombred population than for the average of the three broiler lines (0.25). They concluded that even in lines previously selected for growth there is sufficient variation in efficiency of food utilization to allow progress in selection without further change in body weight.

Responses to 12 generations of single-trait selection for food efficiency and its components were compared by Pym and Nicholls (1979) and Pym (1985). Chickens were selected for increased 5-week to 9-week liveweight gain (line W), increased 5-week to 9-week food consumption (line F), decreased 5-week to 9-week FCR (line E), or at random (line C). Selection for gain resulted in a substantial direct response but also a considerable correlated increase in food consumption, leading to a moderate decrease in FCR. By way of contrast, selection for increased appetite in the F line led to a large direct response, only a moderate correlated response in gain, and as a consequence, a substantial *increase* in FCR. Selection for improved food efficiency in the E line resulted in a moderate correlated increase in gain, essentially no effect upon intake, and consequently a considerable decrease in FCR. Realized heritability estimates calculated from responses to the tenth generation were 0.30 for gain, 0.36 for intake, and 0.26 for FCR; realized genetic correlation estimates were gain:intake +0.64, gain:FCR -0.50, and intake:FCR +0.22 (Pym, 1985).

Sorensen (1984) compared response to four generations of selection for increased 40-day liveweight or reduced 18-day to 40-day FCR on either a normal or a low protein diet. After four generations of selection, 40-day liveweight on the normal diet was about 200 g greater in the high-weight line than in the low-FCR lines, which were similar. Fixed age and fixed liveweight comparisons of FCR measured on the normal diet showed the low-FCR lines to be significantly more efficient than the high-weight line, although the difference was substantially reduced with the weight-constant comparison. The line selected for low FCR on the low protein diet grew no better on this diet than the line selected on the normal diet, whereas the former line tended to grow a little faster than the latter on the normal diet.

Leenstra and Pit (1987) compared the performance of four lines of chickens after four generations of selection for either low abdominal fatness (line AF), low 21-day to 42-day FCR (line FC), and high 42-day weight on *ad libitum* feeding (line GL) or restricted feeding from four to 42 days of age (line GR). Excluding the results of the AF line, 42-day liveweight was 230 and 75 g lighter in the FC and GR lines respectively than in the GL line, whereas FCR to 41 days of age was 0.20 and 0.18 greater in the GR and GL lines respectively than in the FC line. These results indicate that while selection for food conversion efficiency alone does not yield the growth response achieved by selection for growth rate, the relative gains in feed efficiency are substantial. With regard to responses in growth and efficiency, the results further show little benefit from selection for growth under restricted feeding, although it is likely that the method and degree of restriction chosen could have quite profound effects upon the relative response.

Chambers (1987) reported on responses to five generations of multi-trait selection for growth, feed efficiency, and abdominal fat in lines of chickens derived from commercial broiler sire strains. Selection was for high 28-day weight combined with low abdominal fatness (line S1), high 28-day weight combined with low 28-day to 42-day FCR (line S2), and all three traits (line S3). After five generations of selection there was little difference among the three lines in growth rate, but feed efficiency in line S2 appeared to be superior to line S3. Feed efficiency was not measured in line S1. Realized heritabilities measured in the three lines ranged from 0.28 to 0.57 for 28-day liveweight, and from -0.31 to +0.33 for 28-day to 42-day feed efficiency.

There is thus considerable genetic variation in feed efficiency independent of growth rate which the commercial breeder can manipulate. The use of FCR or its inverse as the sole selection trait would not appear to be indicated because of the lower than optimal correlated response in growth rate. There would further appear to be limited value in selecting for feed efficiency under a dietary regime other than that typically used for the commercial meat bird, although evidence here is restricted to only one comparison of diets varying in protein level. Perhaps the most appropriate means of maximizing economic response with respect to growth and food intake is to combine selection for these two traits, using either independent culling levels or an index, as proposed by Pym and James (1979) and Leenstra (1988).

Correlated response in body composition. Since fatty tissue in birds is energetically dense, containing only about ten percent moisture, while protein in muscle is associated with about 70 percent moisture, it would seem axiomatic that selection for improved feed efficiency should result in a reduction in body fatness. Brody (1935) thus predicted that inefficient strains would store less protein and more fat than strains with higher efficiency. Thomas et al. (1958b), in support of this prediction, found that chickens selected from dam-family groups exhibiting low efficiency were fatter than those from corresponding high-efficiency groups.

Selection for improved feed efficiency has been shown to result in a reduction in either total body fat or abdominal fat at a given age (Washburn et al., 1975b; Pym and Solvyns, 1979; Sorensen, 1984; Leenstra and Pit, 1987; Chambers, 1987) or weight (Pym, 1985). The reduction in fat would not appear to be as great as that achieved through sib selection for reduced fatness (Leenstra and Pit, 1987). Selection for increased gain was shown to have little effect on body fatness whether measured at a given age or weight, whereas selection for increased food consumption resulted in a marked increase in fatness (Pym, 1985). As might be predicted from the above selection experiment results, birds from a more efficient commercial broiler line had significantly less abdominal and total body fat than those from a less efficient line (Cahaner et al., 1987).

The reduction in fatness in lines selected for improved feed efficiency has been shown to be associated with a significant decrease in 16:1 and an increase in 18:0 fatty acids (Washburn et al., 1975b), a decrease in adipose cell volume and a decrease in lipogenesis as measured by the activity of NADP-malate dehydrogenase and the incorporation of [U-^{14}C] glucose into radioactive lipid (Hood and Pym, 1982), and a reduction in plasma low-density lipoprotein concentration (Whitehead et al., 1984).

Genetic improvement in feed efficiency is thus likely to be associated with a decrease in body fatness, which in turn is due in part to a reduction in fatty acid synthesis, a decrease in plasma low-density lipoprotein concentration, a decrease in adipose cell volume, and a reduction in the size of the abdominal fat pad. Selection for improved feed efficiency therefore not only improves this most economically important trait, it also serves to reduce body fatness, which is presently the cause of considerable concern to the chicken meat industry.

Effects on energy and nitrogen metabolism. In the experimental lines of Pym and Nicholls (1979) selected for increased growth, appetite or feed efficiency, there were a number of line differences in the components of energy and nitrogen metabolism after three (Pym and Farrell, 1977) and ten (Pym et al., 1984) generations of selection. Line means for some of the component parameters are shown in Table 34.1. Whereas selection for low FCR resulted in an increase in metabolizable energy, net availability of ME for gain (NAME), and nitrogen retention, and a decrease in maintenance energy requirement, selection for high appetite had the opposite effect.

As shown in Table 34.1, protein breakdown measured as N^{t}-methylhistidine excretion rate was significantly lower in line E than in lines C and F (Tomas et al., 1988), and the fractional synthesis rate of protein was significantly higher in line E than line F (Johnson et al., 1986). Selection for improved feed efficiency thus resulted in a significant improvement in protein accretion rate, whereas selection for increased appetite had the opposite effect.

Washburn et al. (1975a) examined differences in ME and nitrogen retention between two previously randombred lines selected for one generation for divergent FCR (Guill and Washburn, 1974) and found no differences in either parameter between the two lines. It is perhaps not unlikely that after such a short period of selection, differences in component physiological traits were not large enough to show statistical significance.

The contribution of feather cover to maintenance requirements is illustrated by a comparison of feathering (Pym, 1985) in the four lines of Pym and Nicholls (1979). Lines W and E were exclusively rapid feathering, line C was segregating for both rapid and slow feathering, whereas the birds in line F were homozygous for delayed feathering, a dominant allele (K^n) of the sex-linked recessive gene (k^+) for rapid feathering (Somes, 1969). These birds were virtually naked until five weeks of age, after which there was a gradual increase in feathering, but differences were still discernable at 15 weeks of age. Selection for high appetite thus favored individuals with poor feathering because of the high energy requirement for body heat maintenance.

Somewhat contrary results were obtained by Cahaner et al. (1987) in a comparison between two commercial broiler sire lines, where birds from the significantly more efficient line had poorer feather cover than those from the less efficient line. In this study, however, reasonably warm temperatures were maintained with hot air brooding and there may have been no benefit in having better feather cover. Differences in activity appear to have contributed in some measure to line differences in feed efficiency.

FOOD UTILIZATION EFFICIENCY FOR EGG PRODUCTION

Most of the selection emphasis in commercial layer lines has been for increased egg

Table 34.1. Components of energy and nitrogen metabolism in lines of chickens selected for increased growth (line W), appetite (line F), or food utilization efficiency (line E), or at random (line C). Standard errors in parentheses.

Line	Metabolizability of dietary energy[1],%		Daily maintenance energy requirement[2], kJ ME/kg W		Net availability of metabolizable energy[2], %		N^t-methyl-histidine excretion [3, 5], μ mole/mole carcass N/day		Fractional rate of protein synthesis[4],%
W	73.0	(0.3)	669	(15)	68	(5)	5.48	(.17)	9.54
F	62.7	(1.8)	861	(14)	76	(4)	6.43	(.21)	8.39
E	76.0	(0.3)	699	(13)	85	(6)	4.99	(.16)	9.56
C	72.7	(0.5)	740	(11)	73	(4)	5.86	(.16)	9.50

[1] Determined in generation 12 (Pym, 1985).

[2] Determined in generation 10 (Pym et al., 1984).

[3] Determined in generation 12 (Tomas et al., 1988).

[4] Determined in generation 12 (Johnson et al., 1986).

[5] A measure of protein breakdown rate.

production, increased egg weight, early sexual maturity, and reduced body weight. Such selection has been demonstrated by experimental results to result in considerable improvement in food utilization efficiency for egg production (Frankham, 1971; Fairfull and Gowe, 1979; Jackson et al., 1986).

The corollary to this is that the efficiency with which birds convert food into eggs can largely be accounted for by the number of eggs produced, the weight of the eggs, the age at sexual maturity, and the body weight and body weight change of the bird over the production period.

There have been numerous studies within lines and between breeds and strains of the proportion of food consumption explained by variation in egg mass, body weight, and body weight change. Arboleda et al. (1976a, 1976b) and Wing and Nordskog (1982a, 1982b) found that egg mass and body weight accounted for 30-60 percent of food consumed. Hagger and Abplanalp (1978) found that egg mass, body weight, change in body weight, and age at first egg explained 52-72 percent of food consumption, and Bentsen (1979) noted that 69 percent of food intake could be attributed to these variables plus mortality. Watanabe et al. (1975), Bentsen (1980, 1983a), Byerly et al. (1980) and Pirchner (1980) showed egg mass, body weight, and change in body weight to account for 70-90 percent of food consumed.

In high producing modern commercial layers, the product of egg numbers and egg weight, or egg mass production, accounts for most of the variation in food intake of laying hens, followed by body weight and change in body weight (Fairfull and Chambers, 1984). In absolute terms, however, maintenance accounts for the major proportion of food consumption (Luiting, 1987), and in lower producing and heavier birds the contribution of maintenance is considerably increased. Joshi et al. (1949) found that approximately 71 percent of the food consumed was used for body maintenance, with only 27 percent used for the production of eggs, in New Hampshire pullets measured over an 18-week period commencing at about eight months of age.

The term 'residual feed consumption' (RFC) was coined by Nordskog et al. (1972) to define the remaining variation in food intake that could not be accounted for by specified variables, normally including egg mass, body weight, change in body weight, sexual maturity, and sometimes other defined factors. A multiple regression equation is usually fitted and its coefficients are used to determine the food requirements from each source (Byerly, 1941; Leeson et al., 1973; McDonald, 1978).

Bordas and Mérat (1984) used such an equation to determine residual food consumption of hens in a selection experiment where lines were selected divergently for this trait. A different approach to improvement in feed efficiency was proposed by Hagger and Abplanalp (1978) who, rather than using selection criteria derived from food intake data adjusted by phenotypic regression, constructed selection indexes which utilized information on both phenotypic and genetic variances and covariances between the different traits.

Physiological Components Of Residual Feed Consumption

As for food utilization efficiency for growth, there are a number of factors which account in different measure for the variation in residual food consumption and thus feed efficiency for egg production. The more readily apparent factors are food wastage, digestibility/metabolizability of dietary nutrients, the net availability of metabolizable energy for both egg production and body weight gain, the composition of the product (eggs) and of the body weight change and maintenance costs as affected by metabolic rate, body composition, insulation against heat loss or gain (feather cover), and activity.

While food wastage is both repeatable (r=0.65) and heritable (h^2=0.13) (Heil and Hartmann, 1980), variation in this trait can be eliminated by the use of well designed and managed feeders. It is further a difficult trait to quantify and obviously one that either requires elimination or accurate measurement in a breeding program for improved food utilization efficiency.

Generally, individual variation in metabolizability of dietary energy in layer-type chickens has been shown to be quite low, with a coefficient of variation of about three percent (Foster, 1968). Bentsen (1983b) and Luiting (unpublished results cited by Luiting, 1987) were unable to find significant correlations between metabolizability and food consumption corrected for body weight and egg production. The genetic correlation between these traits found by Bentsen (1983b) was -0.3, which, although not significant, indicated a positive association between efficiency and metabolizability. Morrison and Leeson (1978) were, however, unable to detect significant differences in metabolizability of dietary energy between four 'efficient' and three 'inefficient' White Leghorn hens of similar body weight sampled as the extremes from a population of 100 birds.

Because of the much greater energetic cost of depositing lipid than albumen, there should be a negative association between feed efficiency and the proportion of yolk in the egg. The ranking of strains for FCE changed when it was expressed in terms of dry egg mass rather than total egg mass (Hagger and Abplanalp, 1978). Jackson et al. (1986) observed a lower proportion of yolk in eggs from lines of birds selected for high egg production, and which were more efficient, than in eggs from their less efficient unselected counterparts. Bentsen (1980) found positive correlations between percentage yolk and FCR, and Heil and Pirchner (1979) found relatively less yolk in eggs from line crosses selected for improved FCE than in those from a related commercial cross. Somewhat contrary results were obtained by Willeke (1980), where percentage yolk was higher in eggs from birds selected for three generations for improved FCE than in the base generation, although the confounding of differences in egg size and time of measurement between the two groups may account for the result.

Components Of Energy Metabolism

Many studies of energy metabolism of laying hens reported in the literature have measured the total heat production (HP) of *ad libitum* fed birds, which includes both maintenance heat production and the heat increment of production, or fasting heat production which is a measure of basal metabolic rate. Significant strain and breed differences in total HP of fed egg-laying hens have been observed in a number of studies (Berman and Snapir, 1965; Farrell, 1975; Lundy et al., 1978; MacLeod et al., 1982; Bentsen, 1983b). An observation common to all these studies was that the light-bodied White Leghorn had a higher total HP, corrected for metabolic body weight, than any of the heavier breeds with which it was compared.

The maintenance requirement of laying hens, as determined by calorimetry studies, has been reviewed by Balnave et al. (1978). There was no consistent relationship between body weight and maintenance requirement expressed in kJ ME/kg$W^{0.75}$/day although the lighter White Leghorn strains had a higher maintenance requirement than the heavier breeds. An intermediate weight White Leghorn strain, however, had the lowest maintenance requirement of all the strains tested.

The published values for total HP and maintenance requirement are of relatively similar magnitude indicating that the heat increment of production contributes only a small proportion of the total HP. Further, because of the relative similarity in magnitude of the variation in maintenance HP and total HP, Luiting (1987) concluded that the heat increment of production contributes little to the total variation between strains, suggesting that the net efficiency of production is unlikely to vary much between strains.

In a study of energy metabolism in White Leghorn (WL) and Rhode Island Red (RIR) hens, Bentsen (1983b) obtained a between-strain standard deviation for maintenance requirement of 69.3 $kJ/kgW^{0.75}/day$ which was of similar magnitude to that for heat production of full-fed laying hens, indicating that a large part of between-strain variation in energetic efficiency of egg production is associated with maintenance requirements.

There have been a number of studies of fasting HP in various strains and breeds of laying hens (Berman and Snapir, 1965; Farrell, 1975; Lundy et al., 1978; Damme et al., 1984), which have generally shown strain/breed differences to be of similar magnitude to the differences in maintenance energy requirements, indicating that differences in heat increment at maintenance feeding accounts for only a small proportion of the overall difference in maintenance requirements (Luiting, 1987).

Three studies have demonstrated considerable variation in total HP between groups of *ad libitum* fed WL hens with similar body weight and egg production but with markedly different food intakes and hence food efficiency for egg production (Morrison and Leeson, 1978; Katle et al., 1984; Luiting, 1986). In all three studies HP was considerably greater in the high-intake (low-efficiency) group.

Other Traits

A number of studies have been conducted to determine the influence of certain anatomical traits on the residual variation in food consumption. Mérat et al. (1980) found that about 20 percent of the variation in RFC was accounted for by 12 such traits, and that the five traits which correlated significantly with RFC accounted for about 17 percent of the variation. The prediction equation took the form:

$$RFC = 0.27\ Fe - 0.2\ aF + 0.17\ Int + 0.18\ Li + 0.07\ Bo$$

where Fe = feather weight, aF = abdominal fat weight, Int = intestinal weight, Li = liver weight, and Bo = bone weight. It is interesting that the coefficients for Fe and aF have signs contrary to that which might be expected; that is, more feather cover would be expected to reduce heat loss and thus be positively associated with FCE, and because of the high energetic cost of deposition, less abdominal fat would also be expected to be associated with an improvement in FCE.

In agreement with the earlier described feathering comparisons between the high appetite and improved feed efficiency broiler lines of Pym (1985), a number of studies have found a positive association between feed efficiency and feather cover both between (Balnave, 1974; Leeson and Morrison, 1978; Luiting, 1986) and within (Damme, 1984) strains.

Selection For Feed Efficiency Of Egg Production

Both FCR and RFC have been shown in a number of studies to be moderately heritable (range 0.16 - 0.52, Bentsen, 1980, 1983b; Wing and Nordskog, 1982a, 1982b) indicating that selection for improved food utilization efficiency for egg production should yield moderate response. A number of selection studies for improved feed efficiency of egg production have yielded quite promising results (Nordskog et al., 1984; Bordas and Mérat, 1984; Pirchner, 1985).

The selection experiment of Nordskog et al. (1984) determined the value of feed consumption records by comparing two selection indices, one (I_1) which included body weight (BW), egg mass (EM) and food consumption (FC), and the other (I_2) which included only BW and EM. Feed efficiency, defined as EM/FC, was approximately 0.02 higher in the lines selected on I_1 than in those selected on I_2. This was due to a greater egg mass production, smaller body weight, and reduced food intake of the former group. Age at first egg increased by about six days in the I_1 groups but the I_2 groups showed no such change.

Bordas and Mérat (1984) selected two lines from a Rhode Island Red base population for either positive or negative residual food consumption with selection on individual deviation from the regression of food intake on body weight and weight gain in males, plus egg mass in females. After seven generations of selection, food conversion for egg production, measured as food intake/egg mass production, was 2.69 and 2.97 in the RFC- and RFC+ females respectively, a relative difference of ten percent. They estimated the heritability for RFC to vary between 0.08 and 0.29 with a mean of about 0.2.

Reciprocal recurrent selection for improved FCE, based on the two reciprocal testcross half-sibs of two White Leghorn lines, yielded a 6-8 percent improvement in FCE over three generations of selection (Pirchner, 1985). The improvement was achieved in both lines by an initial moderate increase in egg numbers which plateaued after one or two generations of selection, and a moderate decrease in body weight. Contrary to expectations and the base population parameter estimates, egg weight also showed a decline.

Food Consumption Information And Selection

Individual measurement of food consumption in a commercial egg breeding program would be an expensive undertaking as a routine measure in all selected lines. A number of studies have been undertaken to determine whether the benefits of including direct food consumption measurements can be essentially achieved by including information on predicted changes in food intake as suggested by Nordskog et al. (1969).

Lee and Nordskog (1975) found similar correlations between net income and two performance indexes derived as multiple regression equations with net income as the dependent variable. One index included egg mass, body weight, mortality, sexual maturity, and food intake, and the other contained all variables except food intake. Food intake thus added nothing to the prediction of net income. In more recent studies (Hagger and Abplanalp, 1978; Wing and Nordskog, 1982a; Pirchner, 1985), in which comparison was made between indexes including either direct information on food intake or predicted information from correlations with the measured traits, there was little difference in predicted response in either FCR or income over feed costs between the two indexes.

In general it can be concluded that measurement of food intake may not be necessary in order to obtain good response in economic efficiency of egg production, provided that reliable estimates of genetic and phenotypic parameters between food intake and the measured traits are available to allow accurate prediction of responses.

GENETIC VARIATION IN APPETITE REGULATION AND FEEDING BEHAVIOR

There is now considerable evidence for substantial genetic variation in both gustatory activity and food intake regulation in chickens. An exhaustive series of studies carried out with the high (H) and low (L) weight lines of Siegel (1962) has provided a good insight into the effects of growth rate selection upon food intake regulation and feeding behavior. Barbato et al. (1980) showed that although meal sizes were similar in the two lines, the number of meals per day of the high weight birds was almost twice that of the low weight birds. Feeding bouts were longer for the high weight birds and the time spent per day in nonconsummatory activity was less. Dunnington et al. (1987) found that the L line chickens were less able to compensate for a 24 h fast than were the H or H x L F_1 birds. This was considered to be due to smaller gastrointestinal tracts (Cherry et al., 1987), general hypophagia (Burkhart et al., 1983), and modified feeding rhythms (Barbato et al., 1982) of the L line chickens which prevented them from compensating fully after 24 h of fasting. The H parental line and the HL crossbreds were shown to have similar food consumption (Barbato et al., 1983).

The observation by Nir et al. (1978) that food consumption of meat-type chickens, but not egg-type chickens, approaches the capacity of the gastrointestinal system is supported by the study of Iskandar (1988) where commercial broiler birds were shown to be incapable of increasing intake in response to reduced dietary nutrient density in unpelleted diets. The lines of Pym and Nicholls (1979) were also tested in the study and the F line selected for high appetite was shown to exhibit a similar response to the broilers, whereas the E line, selected for improved feed efficiency, responded by increasing their intake of the lower nutrient density diets. It would appear that intense long-term selection for increased growth rate places considerable emphasis on appetite, to the extent that the birds are incapable of eating a greater volume of food regardless of energy content. It is not clear what the determining factors are or what their relative contribution is to this limitation in intake of low nutrient density diets in lines intensively selected for increased growth rate. The particular concern with this development is that the bird has a reduced ability to compensate for diets suboptimal in energy, and when presented with such diets growth performance is likely to be significantly reduced.

One mechanism by which this inability of meat-type birds to increase intake may operate has been shown by Burkhart et al. (1983) to be due to a depressed sensitivity of the satiety centre, located in the ventromedial hypothalamus. Lesioning of this region of the hypothalamus has been shown to result in increased food consumption and to induce obesity in egg-type chickens (Lepkovsky and Yasuda, 1966). However, Burkhart et al. (1983) found that electrolytic lesioning of the ventromedial hypothalamus of the 22nd generation birds of Siegel's (1962) H and L lines, produced the expected hyperphagia and obesity in the L line but not in the H line. They suggested that the lack of response in the line H birds suggested absence or improper functioning of satiety mechanisms.

Infusion of glucose into the hepatic portal system has been shown to result in a cessation of eating in layer-type chickens (Shurlock and Forbes, 1981). However, Lacy et al. (1985) observed that following infusion of small volumes of a glucose solution into the hepatic portal system of broiler and White Leghorn chickens, food intake of the White Leghorns was substantially reduced, but it was unaffected in the broilers. This result indicates that selection for growth resulted in either a reduction in sensitivity of the glucose receptors in the liver or an increase in the threshold level. Similar results were reported by Barbato et al. (1982) who showed that birds from Siegel's H line had a higher threshold for detection of both sweet and bitter substances in the drinking water than their L line counterparts.

It would thus appear that while it is possible to decrease the intake of broilers by various means, it is very difficult to effect an increase. This was shown by Barbato et al. (1984) in a study of force feeding with the 23rd generation birds from Siegel's (1962) H and L lines, where the L line birds could be overfed at an earlier age and to a considerably greater extent than their high-weight H line counterparts.

Siegel et al. (1984a) observed that if selection for increased growth resulted in what they termed 'genetic lesions' of the higher neural centres involved with satiety, and if consumptive limits were set mainly by gastrointestinal capacity, then such capacity should be considered in meat-type chicken selection programs, as earlier suggested by Dror et al. (1977). That such selection might be beneficial was indicated by the study of Iskandar (1988) which showed the F line, selected for increased appetite, had a considerably smaller alimentary tract, in terms of the weight and length of the various organ components, than the E line selected for improved feed efficiency. This was in spite of the fact that the daily intake of the former line was about twice that of the latter. The F line achieved its high intake by a considerable increase in passage rate of ingesta and increased frequency of feeding. It would seem, however, that rather than selecting for gut size, the more appropriate method of improving the efficiency of growth and increasing the bird's tolerance to diets suboptimal in energy density is to select for improved food utilization efficiency directly.

GENETIC VARIATION IN NUTRIENT REQUIREMENTS

The requirements of poultry for specific nutrients are generally expressed in terms of the quantity of the nutrient per unit weight of food, per unit ME concentration in the food, or per unit time (day) necessary for optimal growth or egg production. Depending upon how requirements are expressed, strain and species differences can change quite markedly as shown in Table 34.2 for protein requirements. Turkeys had by far the greatest requirement per unit weight or ME content of diet, but on a gram per day basis the requirement for domestic ducks was much greater. As a consequence of these relationships, chickens and turkeys had similar body weight gains per gram of protein consumed (2.2-2.3), whereas in domestic ducks the ratio was considerably larger (2.9) indicating a greater protein utilization efficiency in this species.

Genetic differences in nutrient requirements for egg production as influenced by variation in rate of egg production and body weight are also affected by the method of expressing nutrient requirement. The point is nicely illustrated by the results of Sharpe and Morris (1965) where diets containing graded levels of protein were fed to Rhode Island Red x Light Sussex pullets and to commercial hybrid pullets. The crossbred pullets weighed 33 percent more and produced 20 percent fewer eggs than the hybrid pullets. The protein requirement for maximum egg production, expressed as a percentage of the diet, was 16.5 percent for the hybrid birds and 12.5 percent for the crossbreds. When differences in food consumption were taken into account, however, both groups required about 20 grams of protein per day for maximum egg production.

Sorensen (1985) suggested that there are two components of nutrient requirements which are important. The first component is the minimum level of nutrient producing the optimum response, and the second is a measure of the 'homeostatic capacity' of the individual or strain to tolerate deficiencies or excesses of a nutrient. The two

Table 34.2. Protein requirements of chickens, turkeys and ducks expressed in different ways. (Adapted from Nesheim, 1975).

Bird type	Protein requirement			Gain per protein consumed, g/g
	% of diet	g/MJ ME	g/d	
Broiler chick	23	17.2	11.5	2.3
Egg-type chicken	20	16.5	8	2.2
Turkey	28	24.4	12	2.2
Domestic duck	19	17.9	18	2.9

components are illustrated by a comparison of the dose responses of lines A, B and C shown in Figure 34.1.

The dose responses of the three lines conform to the classical shape, where there is a diminishing increment in growth with increasing concentration of nutrient up to a reasonably well defined asymptote, beyond which further supply of nutrient results in a depression in growth. In making comparisons between lines A and B, the latter line grows slower at lower dietary concentrations of nutrient but faster at higher levels and has a higher requirement for maximum growth. Lines A and C in comparison have similar nutrient requirements, but at low nutrient levels line A shows a much greater growth depression than line C. Line C thus shows a greater homeostatic capacity to tolerate low and high levels of the nutrient. Sorensen (1985) makes the point that in the case of nutrients included in the food at very low levels, it would be sensible to produce birds which can at least tolerate marginal shortages or excesses due to either accidents in formulation or where it is necessary to use low quality foodstuffs. Because of increasing competition between animals and humans for the higher quality foodstuff ingredients, it would indeed be desirable to have birds which can make more efficient use of lower quality materials.

Selection experiments have provided a valuable source of genetic material to study

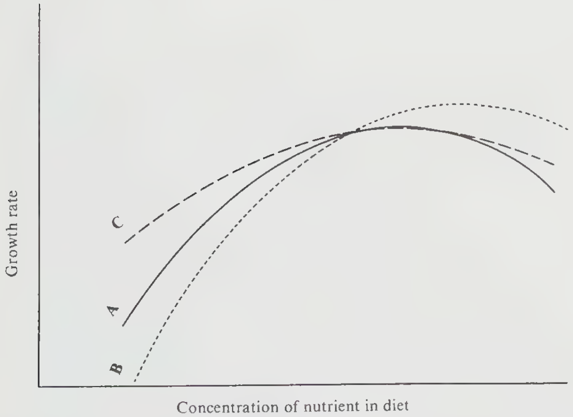

Figure 34.1. Hypothetical dose-response curves for three lines of chickens. (Adapted from Sorensen, 1985).

genotype x diet interactions, and more specifically, genetic variation in nutrient requirements. The classic experiment is one in which selection has been exercised for high and low growth on a 'deficient' diet (and sometimes as well on an 'adequate' diet), and the lines thus generated are subsequently tested for performance on diets ranging in nutrient concentration (see Hutt and Nesheim, 1966 regarding arginine in chickens; Godfrey, 1968 for lysine in Japanese quail). This form of testing gives a very specific indication of genetic variation in nutrient requirements, at least in the stock used, and it is likely to indicate variation if potential for such exists in the base population.

A second type of study is one in which selection has been exercised in different lines for either growth or egg production performance in birds normally given an 'adequate' diet, followed by testing the lines on diets varying in one or more specific nutrients (see Marks et al., 1969; Pilbrow and Morris, 1974; Siegel et al., 1984b). Because selection in this case has generally not been directed at any specific capacity of the birds to either more or less efficiently utilize the nutrient(s) tested, a positive result is less likely than in the first type of study, and a negative result should not be taken as indicating that there is no potential for genetic variation in requirement for the nutrient in question.

EVIDENCE FOR GENETIC VARIATION IN NUTRIENT REQUIREMENTS

Nesheim (1975) gave an excellent and exhaustive review of the then published information on genetic variation in nutrient requirements in poultry, and Sorensen (1985) reported on more recent publications in this area with special reference to requirements for growth. Some of the material presented in these two reports is included here.

In any consideration of nutrient requirements, the nutrients can be essentially classified into five groups: energy, protein and amino acids, vitamins, minerals, and water. These will be discussed in order.

Energy Requirements

Energy assumes a special place among the nutrients since the food intake of both growing (Fisher and Wilson, 1974) and laying (Morris, 1968) birds is largely dependent upon the energy content of the diet. From a study of 34 published reports, Morris (1968) concluded that hens offered diets with different energy levels tend to adjust consumption so as to maintain a similar caloric intake. However the adjustment is imperfect, and energy intake increases on high energy diets. In the case of the growing bird, in a study of the data from 51 experiments in 22 reports, Fisher and Wilson (1974) found an increase in growth rate of about four percent per unit increase in ME from 9 to 16 MJ/kg, with no real indication of an optimum level. It is thus apparent why requirements for the other nutrients are often expressed in relation to dietary energy concentration. While energy requirements may be difficult to define in the classical sense, there may be benefit in producing birds which perform well on low nutrient density (low quality byproduct-type) diets.

The classical selection experiment of Falconer (1960), in which mice were selected for increased growth rate under either a 'high' or 'low' plane of nutrition and subsequently tested in the two environments, showed the low plane-selected line to perform not quite as well on the high plane diet, but to very much outperform the other line, in terms of both growth and feed efficiency, on the low plane diet. The low plane line had a capacity to consume considerably more of the low plane diet, which was the high plane diet diluted with oat hulls. However, in a selection experiment with Japanese quail, in which birds were selected for increased growth rate on either a high or low nutrient density diet, Pym and Solvyns (1981) found no differences between lines in growth, feed efficiency, or body composition on either of the two diets. The low density diet was produced by diluting the high density diet with finely ground rice hulls; all nutrients were thus essentially in the same ratio to energy in the two diets. This result indicates that in the stock used in this study at least, selection in the two environments acted essentially on the same physiological mechanisms to yield similar responses.

Breed differences in growth response to dietary metabolizable energy have been reported by Sibbald and Slinger (1963) and Gardiner (1971) in comparisons between White Leghorns and White Rocks. In both cases the growth performance of the White Rock lines was reduced to a much greater extent than the White Leghorns when given the low energy density diets.

Protein And Amino Acid Requirements

A requirement for protein represents a total nitrogen requirement where all the amino acids are present in adequate amounts and without marked imbalances. As Nesheim (1975) pointed out, the requirement essentially represents the net effect of the many metabolic processes in which amino acids participate. Thus the components of a protein requirement can be exceedingly complex, and while it is likely that there is genetic variation in some of these individual amino acid metabolic processes, the sum of their effects (or the genetic variation in total protein requirement) may not be of the same relative magnitude or even in the same direction as the individual components.

Protein and amino acid requirements for growth. Generally speaking, selection studies in which birds have been selected for high or low growth on diets varying in either total protein or specific amino acid composition have shown significant genotype x dietary protein/amino acid interaction (see Marks and Lepore, 1968; Marks, 1971; Hutt and Nesheim, 1966; Sorensen, 1980). There are some experiments in which significant interactions were not obtained (see Lepore, 1965a), but in such cases selection has generally been relatively short-term. Most studies of the response of different commercial meat-type stocks to variation in dietary protein or amino acid composition have found no genotype x nutrition interaction (see Marks et al., 1969; Siegel et al., 1984b). Since such selection is unlikely to have exerted differential pressure on the ability of the different stocks to digest, metabolize, and utilize protein or the component amino acids, the result is not unexpected.

A study of protein requirements (Lepore and Marks, 1971), in lines of Japanese quail selected for 15 generations for increased live weight at 28 days of age on either a high (28 percent CP) or low (20 percent CP + 0.2 percent thiouracil) protein diet (Marks and Lepore, 1968; Marks, 1971), revealed that the requirement for protein for optimum weight gain was similar for the two lines at about 24 percent. However, the growth rate of the high protein-selected line was ten percent greater than that of the low protein line when both were given a 24 percent protein diet, whereas there was no difference in growth performance between the lines on a 15 percent protein diet. Sorensen (1985) suggested that these results could be interpreted as indicating a greater homeostatic capacity in the low protein-selected line.

Two generations of selection for increased growth rate in chickens from a meat-type randombred population given one of three diets containing 14, 18, or 22 percent crude protein (lines L, M and H respectively), resulted in similar genetic improvement in the M and H lines but little progress in the L line (Marks and Britton, 1978). The increase in the H, M and L lines relative to the control line given the corresponding diet was about 25, 21 and 13 percent respectively, with realized heritability estimates of 0.61, 0.42 and 0.22. While it could be inferred from the study that selection for growth rate in meat-type chickens should not be carried out on low protein diets, the response of the lines to variation in dietary protein was not tested and as such no estimate could be made of protein requirements.

Sorensen (1980, 1985) selected meat-type chickens for increased growth performance on either of two diets varying in lysine and methionine. The low diet contained 70 percent of the methionine and lysine in the high diet. When given the high protein diet, the line selected on that diet marginally outperformed the other line, but when tested on the low protein diet, there was a dramatic reversal in relative growth, with the low protein-selected line growing substantially faster than the high protein line. The measured protein requirements in the H and L lines were 18.4 and 17.2 g CP/MJ ME respectively, showing that protein requirements could be altered by selection.

There has been in recent times some interest in the potential value of free-choice feeding which allows birds to select freely between a protein-rich or energy-rich feed (see Mastika and Cumming, 1981; Brody et al., 1984). To explore the possible ramifications of selection under such a regime, Darden and Marks (1988a) selected Japanese quail for 11 generations for high (H) or low (L) body weight under either a complete (CD) or 'split' (SD) diet. Realized heritabilities were higher in the complete diet lines (0.52 H-CD, 0.47 L-CD) than in the split diet lines (0.36 H-SD, 0.30 L-SD). When the two high weight lines were given the split diet (Darden and Marks, 1988b), there was no difference in energy intake, growth rate, or body fatness, but the H-SD line ate more protein than the H-CD line, indicating that selection on the split diet had imparted no special ability to utilize protein more efficiently.

Amino acid requirements. Breed and strain variation in response to arginine deficiency has been observed in a number of studies (Hegsted et al., 1941; Nesheim and Hutt, 1962). To determine the mode and degree of inheritance of such variation, Hutt and Nesheim (1966) selected two strains of White Leghorn chickens for high or low growth rate on an arginine deficient diet containing casein as the source of protein. Studies with the lines over some years (Nesheim et al., 1967; Nesheim, 1968; Austic and Nesheim, 1970; Wang and Nesheim, 1972; Wang et al., 1973), indicated that the difference in arginine requirement of the high and low requirement strains was due largely to a genetic difference in ability of chicks of the two strains to degrade excesses of lysine. Casein which was used in the low arginine diets contains high levels of lysine. Chicks from the high requirement strain were found to have significantly lower lysine-α-ketoglutarate reductase activity in the liver than those from the low requirement strain (Wang et al., 1973). Thus, although selection was exercised for differences in apparent arginine requirement, the basic metabolic changes occurred in lysine metabolism. Despite this large difference in ability to degrade excessive amounts of lysine, the low arginine requirement strain did not appear to have an elevated lysine requirement (Nesheim et al., 1971). This study highlights the complexity of the interrelationships between the amino acids and the potential problems associated with any endeavor by genetic means to improve the net efficiency of protein utilization by the bird.

Given the very large differences between the arginine-selected lines in their response to high dietary levels of lysine, it might be reasonable to expect that there exists considerable genetic variation in lysine utilization where the nutrient is limiting. While there is evidence of some variation in lysine requirement for growth in chickens (Enos and Moreng, 1965) and Japanese quail (Godfrey, 1968), such variation would appear to be only moderate.

In the study by Enos and Moreng (1965), growth rate of offspring of 16 sire families differed significantly when given a lysine deficient diet. However, four generations of selection for high and low growth rate of egg-type chickens on a lysine deficient diet resulted in relatively little response in either direction, with realized heritability estimates of 0.10 and 0.19 in the high and low selected lines respectively (Enos et al., 1971).

In a selection study of lysine utilization in Japanese quail, Godfrey (1968) selected two lines of birds for increased growth rate on either a lysine deficient (line D) or adequate (line F) diet for ten generations. An unselected control line (line C) was also maintained and was given the lysine adequate diet. Although realized heritabilities in the two lines were zero and three percent in the D and F lines respectively, indicating no measured response in either line, when subsequently tested on the two diets, there was a classical interaction. Growth rate on the adequate diet was 14 percent greater in the F line, but on the deficient diet there was a reversal with growth rate 12 percent greater in the D line. Differences in feed efficiency followed the same trend. The result illustrates the importance of measuring selected lines in all selection environments to ascertain the effectiveness of the different selection regimes. Estimates of response in one environment might prove quite misleading as to response in other environments.

There have been a number of studies of genetic variation in methionine requirements in growing birds. Such studies have doubtless been initiated because in normal diets methionine is either the first or second most limiting amino acid, and any genetic improvement in methionine utilization is likely to have significant economic consequences. McDonald (1957) found that White Leghorn chicks had a higher requirement for dietary methionine than Australorps as measured by growth response. Subsequently (McDonald, 1958) it was shown that the growth rate of the White Leghorns was improved by both methionine and cystine supplementation, whereas growth rate of the Australorps was improved only by cystine supplementation. Miller et al. (1960), in later studies with other flocks of White Leghorns and Australorps, showed that although no difference in methionine requirement could be demonstrated between the two breeds, the conversion of ^{35}S-labelled methionine to cystine was greater for the White Leghorn chicks than for the Australorps. Nesheim (1975) suggested that differences between the breeds in the conversion of methionine to cystine are likely due to differences in rate of feathering and the requirement of cystine for feathering; the Leghorn is a rapid feathering breed whereas the Australorp is slow feathering.

In an endeavor to manipulate the methionine requirement in chickens genetically, Hess et al. (1962) selected Athens-Canadian randombred birds for four generations for either high or low growth rate to three weeks on a methionine deficient diet. Each selected breeder was paired with a breeder of similar eight-week body weight from the other line so that inherent differences in growth performance were not responsible for the divergent response in three-week weight in the two lines. Although there was a marked divergence in three-week weight between the two lines given the methionine deficient diet, when given a methionine adequate diet growth rate was similar in the two lines, demonstrating a differential response to dietary lysine. The actual methionine requirements in the two lines were, however, not measured.

A later study (Lepore, 1965a) used an essentially similar design to that of Hess et al. (1962), except that an additional pair of lines selected for fast and slow growth on a methionine adequate diet (lines FN and SN respectively) were included, and eight-week body weight was not paired. After two generations of selection there was no difference in methionine or protein requirements between the four lines. From paired feeding studies of the fast and slow growth D and N lines, Lepore (1965b) concluded that differences in growth performance were largely due to differences in food intake and not to differences in methionine utilization. Even if potential for such existed, it is unlikely that two generations of selection gave sufficient opportunity for the necessary physiological changes to occur.

Protein and amino acid requirements for egg production. Genetic differences in daily protein requirement can arise from one of two causes. Firstly the rate of egg production and the body weight of the bird as it affects maintenance largely determine the daily protein requirement. There is also potential for variation in the efficiency with which the component amino acids are digested and utilized for egg production and maintenance. Such variation would mean that one strain may require more dietary protein per unit of egg production or body weight than another.

There are a number of early reports of strain/line differences in protein requirement (see Harms and Waldroup, 1962; Moreng et al., 1964; Sharpe and Morris, 1965; Harms et al., 1966; Balloun and Speers, 1969). While these reports could be used to derive the responses to calculated doses of a limiting amino acid if reasonable assumptions were made about the composition of the diets, none provides sufficient evidence to determine satisfactorily whether differences in daily protein requirement are due to differences in rate of egg production and body weight of the stock or to differences in the net utilization of amino acids.

Lysine requirements in eight commercial strains of laying hens were estimated by Pilbrow and Morris (1974) using the nutrient input to egg output relationship described by Fisher et al. (1973), which allows estimation of separate requirements for egg production and body weight maintenance. An analysis of responses obtained during the period of peak production between 36 and 47 weeks of age showed significant differences between stocks in the amount of lysine required per g of egg produced and/or in the amount required per kg of body weight maintained.

A comparison of egg production parameters in hens from ten layer strains given either of two diets varying in both protein and energy revealed significant strain x diet interactions for both body weight and egg production (Liljedahl et al., 1973). There were some large differences in the relative egg production response to the two diets of strains with similar body weights, indicating possible differences in the net utilization of protein for egg production.

Vitamins

Because of their nutritional importance and the cost of dietary supplementation, it is important to know if there is genetic variation in requirements for the different vitamins and what those requirements are in the present commercial strains and breeds. Nesheim (1975) provided an exhaustive coverage of studies of genetic variation in vitamin requirements and the reader is referred to that report for more detail. There have been a number of reports of breed differences in response to thiamine deficiency in which White Leghorns were first found to show lower mortality than Rhode Island Red chicks when given a thiamine deficient diet (Lamoreux and Hutt, 1939), but in a later study to show higher mortality than crossbred heavy breed chicks on a thiamine deficient diet (Thornton, 1960). Both Scrimshaw et al. (1945) and Howes and Hutt (1956) found that White Leghorns laid eggs containing a considerably higher concentration of thiamine than those from a number of heavy breeds.

Because of the then considerable economic importance of the effects of riboflavin deficiency upon early growth, egg production, and hatchability, and the unavailability of synthetic sources, two selection studies were initiated in the 1940s to breed birds with a low riboflavin requirement (Lamoreux and Hutt, 1948; Lerner and Bird, 1948). The former researchers also selected a 'susceptible' strain on the basis of poor growth rate on the riboflavin deficient diet. After five generations of selection in the former study, when given the deficient diet, growth rate in the 'resistant' chicks was 45 percent greater than in the 'susceptible' line, whereas there was essentially no difference in growth rate on the riboflavin adequate diet. While riboflavin requirements were not quantified, the study clearly demonstrated genetic differences in response to riboflavin deficiency. Although less clearly differentiated, similar results were obtained by Lerner and Bird (1948).

Studies have shown that hens carrying a recessive gene (*rd*) in the homozygous state are incapable of synthesizing riboflavin-binding protein (RBP), and as a consequence eggs from these birds will not hatch even when the diet is enriched in riboflavin (Maw, 1954; Boucher et al., 1964; Winter et al., 1967). Breed differences in riboflavin content of eggs have been reported (Jackson et al., 1946; Mayfield et al., 1955), which Nesheim (1975) suggests may be the expression of breed variation in RBP secretion.

A similar result to the above selection study was obtained by Nesheim (1972) where White Leghorn chicks were selected for either high or low growth rate on a nicotinic acid deficient diet. After four generations of selection, growth rate of chicks selected for increased growth was 75 percent greater than in those selected for low growth rate, while mortality was about ten times as great (18.3 vs. 1.9 percent) in the latter group. When given the adequate diet, however, there was no difference in growth rate between the lines. Subsequent studies with these birds indicated that the genetic difference was associated with the relative ability of the strains to convert tryptophan to nicotinic acid (DiLorenzo and Nesheim, 1972).

There are large differences between the various species of poultry in their dietary requirements for vitamin D (National Research Council, 1984). The requirement in any species is contingent upon the dietary levels and balance between calcium and phosphorus. Rhode Island Red and White Sussex chicks were shown to require significantly more vitamin D than White Leghorns (Waldroup et al., 1965). Juneja et al. (1982), using electrophoretic techniques, demonstrated the existence of polymorphic variation of a vitamin D-binding protein in Rhode Island Red and New Hampshire chickens.

Breed and strain differences have been reported for a number of the other vitamins including pantothenic acid, pyridoxin, choline, and vitamins E and A, but few detailed studies have been conducted. Selection for high and low blood carotenoid concentration has been shown to result in significant divergence after four generations of selection (Letendre et al., 1968). The blood levels were 8.9 and 5.8 µg/ml in the high and low lines respectively. No measures were made of vitamin A requirement in the lines. Vitamin A requirements expressed as IU/kg diet would appear to be considerably higher in turkeys, domestic ducks and Japanese quail (4000-5000) than in chickens and geese (1500) (National Research Council, 1984).

Given the apparent positive response to selection for 'resistance' to dietary deficiency of a number of the vitamins, there could be some benefit in producing birds with either lower dietary requirements or a greater homeostatic buffering to dietary vitamin deficiency.

However, given the large number of vitamins, their complex metabolic pathways, their relatively low cost, and the already large number of economically important traits that the commercial breeder must include in both meat and egg breeding programs, it is unlikely that resistance to a specific or general vitamin deficiency could be included as a selection trait in a commercial breeding program other than in exceptional circumstances.

Inorganic Elements

Given the very low cost of supplementation and the generally wide tolerance levels, little work has been undertaken to define the extent of genetic variation in mineral requirements. There have been a number of reports of breed and strain variation in metabolism of some of the elements.

Because of its role in bone and egg shell formation, calcium has received most attention. While the parameters of egg shell thickness and strength have been shown to vary with breed and strain (see Hutt, 1949; Nagai and Gowe, 1969), such variation has not been clearly shown to be associated with differences in calcium requirements once differences in intake and rate of production are taken into account (Nesheim, 1975).

In keeping with the higher vitamin D requirements in heavy than in light breeds described earlier, Gardiner (1971) showed that broiler-type chicks had a higher requirement for phosphorus than White Leghorns, whereas Lillie et al. (1964) were unable to demonstrate any difference in phosphorus requirement between two broiler strains.

Differences in requirement for manganese in the prevention of perosis have been reported both within (Serfontein and Payne, 1934) and between breeds and strains (Gallup and Norris, 1939). Breed and strain differences in the effect of this element on hatchability have been reported (Golding et al., 1940; Bolton, 1957).

A genetic component in the response of chickens to selenium deficiency has recently been indicated in a number of studies. Bunk and Combs (1981) found considerable variation between individual Leghorn chicks in growth rate and survival when given a diet containing very low levels of selenium (<0.1 ppm). Similar observations were made by LaVorgna and Combs (1983), who determined that an impairment in the metabolism of the sulphur-containing amino acids may be the site of hereditary involvement in the metabolic requirement for selenium. Halpin and Baker (1984) found similar evidence of aberrant sulphur-amino acid metabolism in a meat-type strain of chickens but not in a strain of White Leghorns. Cunningham et al. (1987) selected a randombred population of chickens for high or low growth rate on a selenium deficient diet for three generations. Significant divergence was obtained with a difference between the two lines in 21-day body weight of 36 g which represented a relative difference of 80 percent. The lines were not tested on diets varying in selenium concentration which precluded an estimate of selenium requirements.

Water

Genetic differences in intake and output of water have been reported in a number of studies. Quisenberry and Malik (1963) found substantial differences in water content of droppings among 30 strains entered in the Texas Random Sample Laying Test. Dunson and Buss (1968) established an inbred strain of chickens which consumed and excreted excessive amounts of water but in all other respects appeared normal. Relative to body weight this strain consumed and excreted more than three times as much water as normal birds of the same type. Studies indicated that the condition was caused by a single autosomal recessive gene (Dunson et al., 1972) but the physiological mechanism controlling the polydipsia in these birds has not been clearly defined.

Selection for increased growth rate has been shown to be associated with an increased intake of water relative to food when water is provided *ad libitum* (Marks, 1980). When food was restricted, water intake remained unchanged, but when water was restricted, food intake was proportionately reduced. Further studies (Marks and Brody, 1984) showed that body weight was depressed more in food-restricted and water-restricted birds from high-weight selected than from unselected chickens. Unselected birds had a greater ability to compensate for food deprivation but there was no difference between the lines in ability to compensate for water deprivation. Selection of normal and dwarf chickens for increased growth rate on normal (0.4 percent) or high (1.6 percent) salt diets for four generations resulted in higher realized heritabilities and gains in the lines given the 0.4 percent salt diets (Marks, 1987). Proportional responses relative to unselected control lines were greater in the dwarf than in the normal birds. When tested on the 0.4 percent salt diet, growth rates and abdominal fatness in the line selected on that diet were greater than in the high salt-selected line.

The studies by Marks and others cited above, have shed new light on the importance of water:food ratios in determining response in growth performance and body composition in meat-type birds, and they provide a possible alternative to the commercial breeder in his endeavors to optimize response in these two most important traits.

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Chapter 35. BEHAVIORAL GENETICS

P. B. Siegel and E.A. Dunnington

For thousands of years humans have been interested in the wide range of behaviors exhibited by poultry (Smith and Daniel, 1975) and behavioral genetics has greatly influenced poultry husbandry and breeding. Initial domestication of species discussed in this book was possible because they exhibited characteristics favorable to the process. These characteristics included: a hierarchical group structure in which sexes could be intermingled and population numbers varied, sexual behavior with males dominant to females and mating signals provided by posture, precocial young with early parent-offspring attachment, nonspecific dietary requirements, willingness to interact positively with humans, and adaptability to a wide range of environments (Hale, 1969). Domestication is a continuing process that involves adaptation of animals to environmental circumstances defined by humans and is manifested by expression of those traits which collectively enable the adaptation (Kohane and Parsons, 1988). When animals are transferred from one setting to another phenotypic expression of some behaviors may change. Species such as Japanese quail, which are monogamous in natural settings, are promiscuous in laboratory and farm settings (Kovach, 1974). Semidomesticated mallard ducks have longer breeding seasons and incubating periods than wild mallards (Prince et al., 1970; Cheng et al., 1980a), suggesting plasticity and stabilizing selection for these traits.

The relationship between behavior and genetics includes two major areas: genetic analyses of behavioral traits and behavioral influences on gene pools of subsequent generations. These areas influence poultry breeding simultaneously. Darwin (1875) suggested that domestic animals were modified through selection and Zeuner (1963) described changes in emphasis of breeding programs during the domestication process. Humans practiced artificial selection by retaining individuals they felt exhibited more desirable traits and/or culling those with less desirable traits. Thus, populations were developed using selection before cell division and Mendelian laws were described or understood. For traits with genetic variation, responses to selection occurred and changes in population means were permanent. Examples include selection for aggressive and against submissive behavior in fighting cocks (Fennell, 1945; Rupert, 1949) and duration of crows. In the latter case, crows of the Totenko fowl last for 20 to 30 seconds (Masui, 1956) in contrast to 2- to 3-second crows for domestic chickens (Siegel et al., 1965).

Behavior-genetic analyses (Siegel, 1970, 1975, 1979) require awareness that, barring mutation, the genotype of an individual is fixed at fertilization and genetic variation is measured in populations. In contrast, behaviors of individuals and of populations (the phenotypes) vary. Social interactions, prior experiences, and genotype x environment interactions all introduce additional sources of variation. Definition and measurement of behaviors are sometimes difficult, but can be quantified if traits are recurrently identifiable and classifiable. As with other traits the phenotypic expression of a behavior is based on genetic background, environment and the interplay of heredity and environment. Qualitative and quantitative procedures for genetic analyses of behaviors are the same as for morphological and physiological traits.

EFFECTS OF BEHAVIOR ON FUTURE GENE POOLS

Differential reproduction is common in poultry. Rate of egg production, fertility, hatchability, and viability are some of the factors which contribute to differences in reproductive capabilities among individuals. Although sexual, incubation, and brooding behaviors also influence reproductive success, the importance of these behaviors in the context of poultry breeding differs depending on the situation. For example, courtship and mating behaviors are no longer of importance in commercial turkey operations where selection for increased breast width has resulted in mechanical barriers to natural mating and made artificial insemination a necessity. Incubation behavior, while essential in the wild, is unnecessary when eggs are artificially incubated. Broodiness has been essentially eliminated through selection in the chicken and Japanese quail by elevating the threshold for expression of the trait (Orcutt and Orcutt, 1976), but elimination of this trait in turkeys continues to be a challenge. Price (1984) has provided numerous examples where domestication has elevated the threshold of behaviors.

Mating behavior, which involves stimulus-response sequences between males and females, has been an active field of study. The theory of sexual selection (Darwin, 1871) attempts to explain evolution of sexually dimorphic traits by addressing differential ability of individuals to acquire mates. According to Wilson (1975), sexual selection consists of epigamic selection, based on choices made between males and females, and intrasexual selection, based on competition between members of the same sex. Mate choice involves both preferences and compatibilities, and may be characterized as a balance between positive and negative assortative mating. Consistent with Wilson's (1975) definition, preferential mating in poultry results from specific male-female interactions with intrasex (male-male and female-female) relationships also having contributing influences. Because of inter- and intrasexual influences, patterns may differ depending on size of flock and male-to-female ratios.

The close relationship between aggressive and courtship behaviors (Siegel, 1983) and the magnitude of differences in dimorphic traits contribute to the complexity of evaluating mate choice. In chickens, small differences in body weight do not result in preferential mating (Lill, 1966; Lill and Wood-Gush, 1965). When differences in body weight are large, however, there is positive assortative mating with preferences depending on the direction of dimorphism (Blohowiak et al., 1980). Large males and females, although generally having low libido, prefer each other. Small males frequently court large females, but mating is rare. In contrast, there is little interest between large males and small females when the weight dimorphism exceeds threefold.

When weight extremes of chickens are not great, there can be negative assortative mating involving social rank with males in higher and females in lower social positions mating most frequently (Guhl et al., 1945; Guhl and Warren, 1946; McDaniel and Craig, 1959). The relationship, however, is equivocal because preference is not expressed when males are tested singly (Guhl et al., 1945; Guhl and Warren, 1946), implying that responses of males are influenced by flockmates. A socially castrated male in a multiple-male flock will mate when alone with females (Guhl, 1949), which may reflect passive dominance of males over females. The social environment within each flock will influence both direction and degree of assortative mating due to dominant-subordinate relationships. In some small flocks, males in higher social positions spend proportionately more of their efforts on intra-sexual aggression than on sexual behavior (Siegel, 1959) while in other flocks, they do most of the mating (Siegel, 1959; Craig and Bhagwat, 1974). When domestic chickens are returned to the wild, the subordinate male of a pair has been observed to do most of the mating in a few cases (Duncan et al., 1978), and in large flocks there is little effect of social rank on mating frequency (Craig et al., 1977). Frequency of mating by an individual, however, does not imply a lack of mate choice in chickens (Blohowiak et al., 1980) or Japanese quail (Blohowiak and Siegel, 1985).

Sociobiology, the systematic study of the biological basis of all social behavior (Wilson, 1975), has generated considerable interest in the biological community on mate selection in the context of optimization between inbreeding and outbreeding. Mate choice is influenced by genetic and learning components. Wild and game farm mallard males raised with ducks of their own strain court females of their own strain more than those of the other strain. When raised with females of the other strain, drakes courted ducks of both strains with equal intensity (Cheng et al., 1979). Females of both wild and game farm strains paired with males raised with members of their strain regardless of male's strain and her rearing experience (Cheng et al., 1978, 1979).

Plumage coloration, an easily identifiable and frequent polymorphic genetic variant, has been used to study preferential mating in poultry. Polymorphisms for plumage coloration cause stratification in the social hierarchies of turkeys (Hale and Buss, 1960) and in the Fayoumi fowl, resulting in nonrandomness of mating (Crawford and Smyth, 1964; Sefton and Crawford, 1967; Crawford, 1973).

Several studies of genetic morphs and learning components on mate choice have been conducted with Japanese quail (eg. Gallagher, 1978; Bateson, 1983; Blohowiak and Siegel, 1983a, 1985). They show that quail distinguish among individuals and exhibit preferences based on plumage color. When choices were made among wild-type and plumage mutants albino and yellow, albino females were avoided whether or not males had previous experience with albinos. No distinction was made between wild-type and yellow. Regardless of whether wild-type, redheads and albinos were reared in separate flocks or intermingled, redhead and wild-type males generally avoided albino females but albino males showed no preference. For females, however, redheads and wild-types raised with albino males exhibited no preference with regard to male morph. These experiments show that experience with plumage morphs appears to finetune the genetic mechanisms underlying mate choice and that responses differ for the sexes.

The head and its appendages are important for individual and group recognition in chickens (Guhl and Ortman, 1953; Candland, 1969). In flocks where there are different comb types, chickens with single combs assume the higher social rank (Siegel and Dudley, 1963; Williams et al., 1977) and males mate more frequently (Crawford and Smyth, 1965). High social rank also confers greater access to feeding and watering areas (Banks et al., 1979) which may be reflected by increased growth and productivity. During development of the commercial broiler industry, synthetic male lines were developed which included segregation at the rose and pea comb loci. Selection for body weight resulted in changes in gene frequencies at these loci with increases in the homozygous recessive single comb morph. Greater access to feeding areas because of social rank may have resulted in single comb chickens being heavier and being selected as breeders for producing the next generation. This artifact in selection, in combination with greater access to females in breeder flocks, could explain the preponderance of single comb chickens in broiler flocks. Although informative in terms of general behavior, interest in effects of plumage and comb morphs on reproduction in commercial poultry breeding flocks has decreased in recent years because stocks are generally of white plumage and chickens are usually single-combed.

The above discussion demonstrates the complexity of behaviors influencing gene frequencies in subsequent generations. Results from the wild and in small flocks may or may not be relevant to commercial poultry operations and inconsistencies in results imply a need for further research in this area of poultry science.

SPECIFIC LOCI AND BEHAVIORS

Numerous morphological, physiological, immunological and behavioral phenotypes can be traced to the presence or absence of specific alleles. Although detailed chromosomal mapping is generally lacking for poultry, data on linkage groups are accumulating, particularly for the chicken (see Chapter 21). Alleles generally have more than one effect and those influencing behaviors are usually classified as to whether their effects are indirect or direct.

Indirect effects are those that modify morphological appearance and/or metabolic needs, resulting in changes in behavioral variation. Plumage color and head appendages discussed in the previous section are examples. Others include autosomal recessive blindness and diabetes insipidus. Blind males will court, but are unsuccessful in completing the mating act (Cheng et al., 1980b). With continuous exposure to females, however, some blind males are successful with practice (Ali and Cheng, 1985). Chickens with diabetes insipidus, because of reabsorption failure in the kidney, exhibit polydipsia (Benoff and Buss, 1976).

When the primary influence of an allele is on the behavior, the effect is considered direct. Numerous examples exist for chickens and are described as abnormalities caused by mutants. The word mutant implies alteration of a certain function of the nonmutated allele. Most behavioral mutants reduce reproductive fitness, have a degree of lethality, are recessive and are frequently located on the Z chromosome. Behavioral mutants inherited as sex-linked recessives include cerebellar hypoplasia (Markson et al., 1959), sex-linked lethal (Goodwin et al., 1950), gasper (Price et al., 1966), jittery (Bohren, 1950), paroxysm (Cole, 1961), and shaker (Scott et al., 1950). Autosomal recessive mutants include congenital loco or stargazer (Knowlton, 1929), congenital tremor (Hutt and Child, 1934; Okano, 1980), congenital quiver (Smyth et al., 1985), crazy (McGibbon, 1973), epileptiform seizures (Crawford, 1970), pirouette (McGibbon, 1974), faded shaker (Silversides and Smyth, 1986) and tipsy (Hawkes et al., 1973).

Although most behavioral mutants influence the nervous system, little is known about pathways which cause the phenotypic changes. Some, such as congenital loco, have been described not only for chickens but for turkeys (Cole, 1957) and Japanese quail (Sittman et al., 1965; Savage and Collins, 1972). Genetic background and environmental factors can also influence expression. Incidence of congenital ataxia was increased by selection (Bernier et al., 1975a) and by mineral deficiencies in the diet of the breeder hen (Caskey et al., 1944; Bernier et al., 1975b). Exposure to intense light in the visible spectrum precludes phenotypic expression of bobber, the sex-linked recessive cervical ataxia mutant in turkeys (Harper et al., 1988). That paper contains several pictures of the phenotypic expression of ataxia.

The mutant most intensively studied from its behavioral and physiological expression is epileptiform seizures (see reviews by Crawford, 1983; Crichlow, 1983; Johnson and Davis, 1983; Corner and Romijn, 1983). Variation in severity of epilepsy among individuals may be attributed to a combination of background genome, environmental factors, and age effects. Because of similar expression of several neurological mutants (Ookawa, 1977), these reviews provide important insights for studying pathways by which behavioral mutants cause phenotypic changes in poultry.

QUANTITATIVE ANALYSIS OF BEHAVIORAL TRAITS

> "We can ask what differences exist between genetic programmes responsible for behaviours formerly called innate and those considered experimentally acquired. A genetic programme which does not allow appreciable modifications during the process of translation into the phenotype I call a closed programme - closed because nothing can be inserted in it through experience. Such closed programmes are widespread among the so-called lower animals. A genetic programme which allows for additional input during the lifespan of its owner I call an open programme."
>
> Mayr (1974)

Quantitative genetics is concerned with inheritance of differences among individuals on a continuous rather than discrete scale. Allelic differences at many loci are assumed and analyses attempt to separate phenotypic variation into genetic and nongenetic components. When joint effects of heredity and environment are not additive, as is frequently the case for behavioral traits, there are genotype x environment interactions. Thus, quantitative genetic analysis measures the degree to which a genetic program is open.

Differences in behaviors among breeds and among strains within breeds of poultry are common and include traits such as perching (Faure and Jones, 1982), crowing duration (Ichinoe et al., 1974) and tonic immobility (Jones and Faure, 1981). Although these differences provide presumptive evidence of genetic variation, they do not address genetic variation within populations or explain the genetic basis for a behavior.

Primary tools for measuring genetic variation of quantitatively inherited behaviors are selection, crossing comparisons among strains, and estimation of heritabilities. These procedures are used in genetic analysis of behaviors *per se* and in measuring influences of behaviors on other traits. In the latter case, the focus may be to use behaviors as an indirect measure for other traits. Examples include genetic variation for feed consumption which influences feed efficiency (Guill and Washburn, 1974; Pym, 1985; Klemm and Pingel, 1987) and plasma corticosterone response to social strife resulting from an unstable social hierarchy (Gross et al., 1984; Gross and Siegel, 1985; Siegel, 1989).

In the latter case we were interested in evaluating heritable differences in responses to social strife. White Leghorn chicks were reared as sex-intermingled flocks in battery brooders to about 35 days of age, when they were transferred to other cages and maintained as 8-bird groups. At about 63 days of age, they were subjected to social strife by moving them into new unisexual flocks on a routine basis for 14 days, avoiding contact with previously encountered individuals. Plasma corticosterone concentrations were then measured. Opposite extremes from the base generation provided parents to produce lines where truncation selection was practiced for either high or low plasma corticosterone concentrations. Separation of lines was rapid and, by generation 6, overlap in plasma corticosterone distributions between individuals of the selected lines was small (Figure 35.1). Because plasma corticosterone concentrations were similar for both lines prior to rotation, differences between lines were due to the social strife. In generations 7 and 8, the selection criterion was changed from plasma corticosterone concentrations to ratios of heterophils to lymphocytes in peripheral blood after two days of rotation (Gross and Siegel, 1985). Differences between lines for heterophil to lymphocyte ratios were similar to those for plasma corticosterone concentrations, being greater for the high than low line with essentially no overlap between lines.

When chickens from these lines were fasted for two days or given an intravenous inoculation of *Escherichia coli* (nonsocial stressors), both lines responded similarly. These

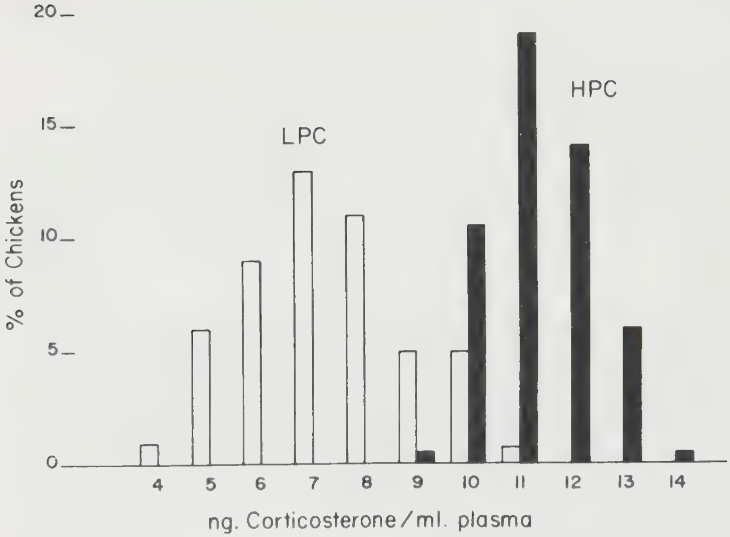

Figure 35.1 Distributions of plasma corticosterone levels for lines of chickens selected for six generations for high (HPC) and low (LPC) plasma corticosterone levels response to social strife. (From Gross et al., 1984).

results implied that selection was for differences in levels of perception of environmental situations or in behavioral adaptations to the social stressors and that the low line could filter out physiological processes associated with the General Adaptation Syndrome (McBride, 1980; Gross et al., 1984; Siegel, 1984). The results also demonstrate that genetic variation in perception of environment can preclude effects of the General Adaptation Syndrome.

Heritabilities have been estimated for numerous behaviors in poultry. Examples include .11 for broodiness (Saeki, 1957), .09 for imprinting (Fischer, 1969), .58 for tonic immobility (Gallup, 1974), .03, .15, .23 and .23 for red, yellow, green and blue color preferences, respectively (Hurnik et al., 1977), .18 for locomotor activity (Jezierski and Bessei, 1978), .09 for duration of prelay restlessness (Heil, 1984), and .07 for feather pecking (Bessei, 1985). Difficulties with generalizations of heritability estimates are that they are population-specific and although low estimates suggest fitness traits, moderate to high estimates could result from selection for an intermediate optimum.

There is an additional difficulty in making inferences about behaviors where measurement is narrowly defined for the test situation, such as with imprinting. For behaviors such as broodiness, there is a threshold for an all-or-none expression which can occur periodically throughout life. For others, the processes are not continuous and may be influenced by diurnal rhythms, such as feeding behavior and food consumption (Masic et al., 1974). Regardless of these caveats, responses to selection have been demonstrated for broodiness (Goodale et al., 1920), imprinting (Graves and Siegel, 1969), and feed consumption (Pym and Nicholls, 1979).

For imprinting, response through five generations of selection was asymmetrical and realized heritabilities were .28 in the fast and essentially zero in the slow line (Graves and Siegel, 1969). Goodale et al. (1920) reduced broodiness from over 90 percent to less than

20 percent in five years of selection in White Leghorns. Also in five generations of selection, change in feed consumption from five to nine weeks of age was approximately 500 g with a realized heritability of .44 (Pym and Nicholls, 1979). These examples show that quantitative genetic analyses of behaviors can be conducted as they are for morphological and physiological traits.

RESPONSES TO SELECTION

Single-trait selection experiments have been conducted for an array of behaviors in poultry (Bessei, 1984). Those by Guhl et al. (1960) and Craig et al. (1965) for relative aggressiveness provided the basis for many subsequent studies. Using randombred populations of White Leghorns and Rhode Island Reds, Craig et al. (1965) selected bidirectionally for high and low social dominance scores of males in initial paired contests. Both randombred base populations were maintained as unselected controls for their respective lines. Five generations of selection resulted in large differences between the selected lines and their respective controls, implying considerable additive genetic variation for the selected trait. Also, the response was neither age nor sex specific.

Examples of other selection experiments for behaviors in poultry include male mating behavior (Wood-Gush, 1960; Tindell and Arze, 1965), imprinting (Graves and Siegel, 1969), tonic immobility (Gallup, 1974), floor laying (McGibbon, 1976), color preferences (Hurnik et al., 1977), open field activity (Faure 1975, 1981), locomotive activity (Bessei, 1979; Jones et al., 1982), and dustbathing (Gerkin and Peterson, 1987; Gerkin et al., 1988). In most cases responses to bidirectional selection were asymmetrical and resulted in changes in unselected traits. None of these experiments were long-term, and information on selection limits and long-term fitness effects are generally lacking.

Perhaps the most detailed discussion of long-term selection and underlying mechanisms is that for perceptual preferences in Japanese quail (Kovach, 1986). Visual discrimination for color and design was assessed and lines were developed that were more than three standard deviations removed from the control line. These populations have been employed in a series of investigations to explain not only behavioral attributes but also physiological mechanisms associated with vision.

Long-term bidirectional experiments for mating frequency in male chickens (Bernon and Siegel, 1983a; Dunnington and Siegel, 1983) and Japanese quail (Bernon and Siegel, 1983b; Blohowiak and Siegel, 1983b) have been conducted. In both species, the base population was maintained as an unselected control and, in the quail, selected lines were replicated. General patterns of response were similar for both chickens and quail (Figure 35.2). Response was asymmetrical (greater in the high than low lines), small during the first few generations, and then greater with periodic increases. The low lines diverged into maters and nonmaters. That is, selection for low mating frequency caused males to pass a threshold below which they would not mate naturally within a limited time period. This effect was not because of a lack of circulating testosterone, suggesting differences at the receptor level. Mating frequencies of crosses between parental lines were significantly higher than midparent values indicating a heterotic effect which lowered the threshold for mating activity. Selection for mating frequency of males did not result in correlated responses of behaviors with production traits for females.

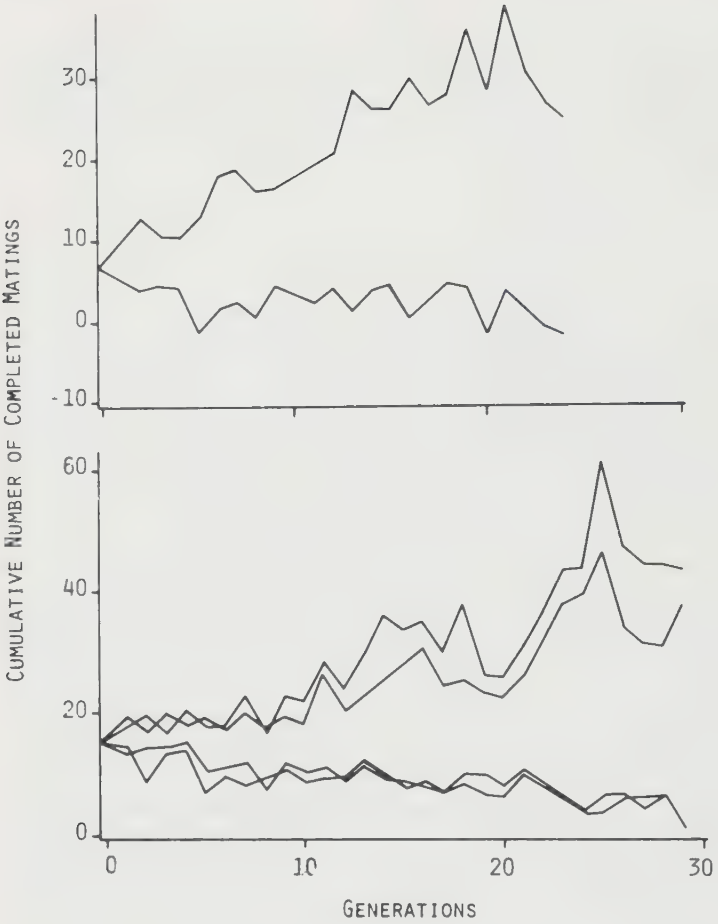

Figure 35.2. Cumulative number of completed matings (control-adjusted) by generations for lines of chickens (top) and Japanese quail (bottom) selected for mating frequency of males. (Adapted from Blohowiak and Siegel, 1983b, and from Dunnington and Siegel, 1983).

CORRELATED RESPONSES OF BEHAVIORS TO SELECTION

Changes in behaviors have occurred during the development of commercial poultry stocks and husbandry practices. Behavioral and genetic adaptations of commercial egg stocks, during the rapid transition to high density environments, has been thoughtfully reviewed by Craig (1982). His review provides support for the thesis of Kohane and Parsons (1988) that genetic variance is both substantial and largely additive when animals are shifted to a new environmental setting, and that behavioral changes are rapid with domestication. The implication is that behavioral plasticity in the new environment facilitates closer association between genotypic and phenotypic values.

The poultry meat industry provides an example of behavioral changes concurrent to selection for greater body size. Selection for increased body weight results in correlated responses in feed intake (eg. Lepore, 1965; Pym and Nicholls, 1979; McCarthy and Siegel, 1983; Klemm and Pingel, 1987) and consumption is approaching gut capacity for commercial meat stocks (Nir et al., 1978). Overconsumption of food causes obesity and reproductive complications in breeders. To alleviate these complications, husbandry procedures to restrict food consumption are routine in poultry meat production (eg. McDaniel, 1983; Robbins et al., 1986; Katanbaf et al., 1989).

Behavioral components of food regulation systems of poultry were reviewed in an excellent symposium edited by Boorman and Freeman (1979). More recently, studies of behavioral-genetic aspects of food consumption of poultry have been summarized by Siegel et al. (1984) and Denbow (1985). There are large genetic components for food intake and for water intake at hatching (Marks, 1980, 1981; Barbato et al., 1983). Although fed complete diets, genetic variation exists among and within populations for balancing diets when nutrients are presented in solid or liquid form (Huey et al., 1982; Gidlewski et al., 1982; Brody et al., 1984). In a bidirectional experiment, Japanese quail were selected for 4-week body weight in an environment where they could self-select dietary protein and energy cafeteria style or in an environment where they were provided a single-source diet (Darden and Marks, 1988a). Responses to selection through 11 generations were similar regardless of feeding environment. Increases in weight over the control line were 49 and 50 percent for the cafeteria and single-source regimes, respectively. Values for the low-weight lines were 46 and 45 percent. Realized heritabilities for body weight, however, were lower with cafeteria feeding (.33 vs. .50, respectively) and selection differentials were greater resulting in the similar response to the two feeding regimes.

Lines of chickens bidirectionally selected for 56-day body weight (Dunnington and Siegel, 1985) differ in food intake behavior (Dunnington et al., 1987). Both lines consume food in discrete meals of about 6.7 g; however, the high line consumes approximately twice as many meals (14) as the low line (8) per day. Duration of feeding bouts are about 13 min for the high line and 8 min for the low line. Nonconsuming feeding behavior in a 24-h period was only 297 min for the high line, but 450 min for the low line (Barbato et al., 1980). Comparisons of normal and dwarf chickens within each line show that the primary influence on feeding behavior is background genome rather than alleles at the *dw* locus. Correlations of meal size with pre- and post-meal intervals suggest involvement of both meal activation and termination mechanisms. Electrolytic lesioning of the ventramedial hypothalamus (Burkhart et al., 1983) produced expected overconsumption of food and obesity in the low weight but not in the high weight line hens. This finding suggests that long-term continuous selection for high 56-day body weight resulted in 'genetic lesions' causing improper functioning of brain satiety centres.

Overconsumption or underconsumption of food from a single source does not imply difference in ability to discriminate among food sources. Barbato et al. (1982) allowed males from the selected lines and reciprocal F_1 crosses to display preferences for dextrose or for quinine sulfate solutions in choice comparisons. Choices were between water and either of the test solutions with the measure being a preference index of volume of water to the test solution. No differences between populations were found at either the lowest or highest concentrations of dextrose or quinine sulfate (Figure 35.3). At intermediate levels the pattern for dextrose mirrored that for quinine sulfate, i.e. preference for dextrose and aversion to quinine sulfate. Comparisons between parental lines and F_1 crosses showed nonadditive genetic variation for sensitivity toward these compounds. The data also showed that while discrimination was evident in all populations, the line selected for heavier body weight had higher thresholds (less sensitivity) than the line selected for low body weight.

In poultry, as well as other higher vertebrates, there are age and body weight minimums that are population-specific, as well as carcass composition requirements for the onset of sexual maturity (eg. Bornstein et al., 1984; Zelenka et al., 1984). In the low-weight line discussed above, some females are anorexic - they eat so little food *ad libitum* that onset of lay is greatly delayed or prevented (Dunnington et al., 1984). When forcefed these females will commence lay (Zelenka et al., 1988). This syndrome has had an effect on the response to selection in these lines (Siegel and Dunnington, 1987). Although gradual delay in sexual maturity occurred concomitant to selection for low 56-day body weight, a threshold was reached when over 50 percent of the pullets did not mature in 1982 and 1983, i.e. generations 25 and 26 (Figure 35.4). At selection age (56 days), females that matured were heavier than those that did not, so selection pressure was reversed. Body weights in generation 27 exceeded those in generation 26 and 83 percent of the pullets commenced lay. Since then the pattern has become cyclical.

The examples provided in this section show that genetic variation exists for a wide array of behaviors. When recurrently identifiable and classifiable, these behaviors respond to artificial selection. More importantly, in the context of applied poultry breeding, correlated responses in behavior which occur with selection for production traits may be favorable or unfavorable in an economic sense.

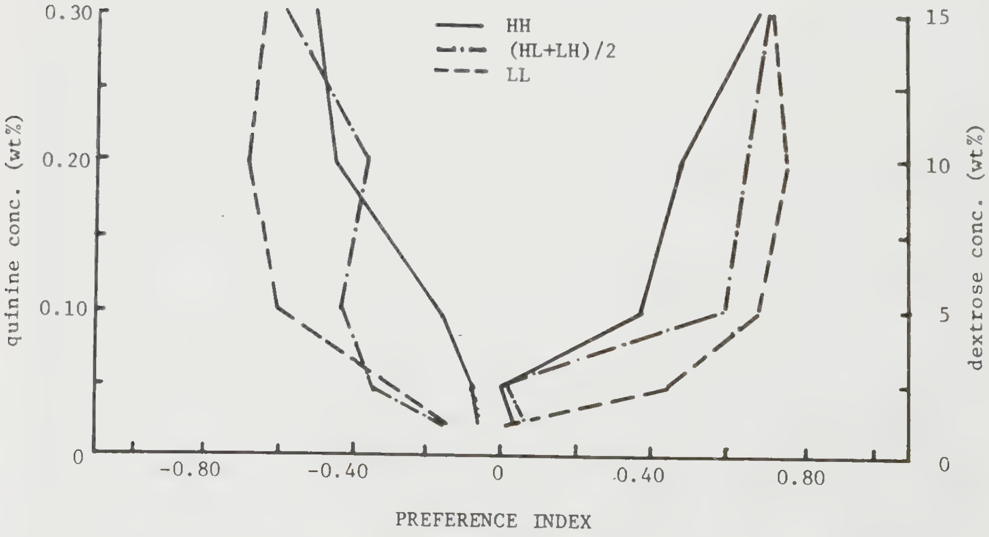

Figure 35.3. Sensory landscape of preference index for dextrose and for quinine sulfate for lines of chickens selected for high (HH) and low (LL) body weight and the mean of reciprocal crosses between them. (From Barbato et al., 1982).

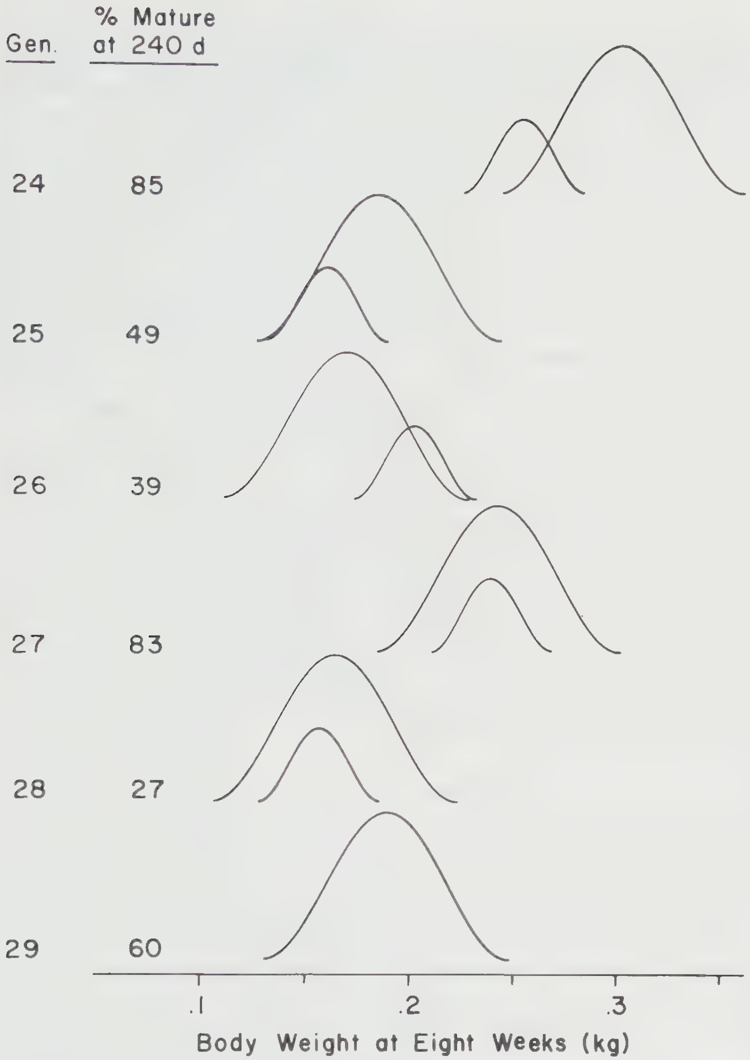

Figure 35.4. Population means (larger curves) and means of selected parents (smaller curves) of females from a line of chickens selected for low body weight with some individuals being anorexic. (From Siegel and Dunnington, 1987).

GENOTYPE x ENVIRONMENT INTERACTIONS

Genotype x environment interactions occur when, relative to each other, a series of genotypes respond differently in a series of environments. Although basic models of quantitative genetics assume no genotype x environment interactions, their importance is well recognized in poultry breeding as evidenced by a chapter devoted to the topic in this book. Moreover, quantitative geneticists have long had an interest in the role of behavior in genotype x environment interactions (eg. McBride, 1958; Dickerson, 1962). Presence of genotype x environment interactions not only limits inferences of how different genotypes respond in various environments, but they also influence estimation of breeding values. This can be important particularly in interpreting data when there have been dramatic changes in husbandry such as that seen with high density housing of layers (Dickerson and Mather, 1976) and in developing breeding programs for these environments (Craig, 1984).

Earlier, we discussed possible effects of social stratification on randomness in mate choice, and estimation of breeding values. Strain comparisons show higher productivity of pullets from aggressive strains when housed singly or intermingled in groups with less aggressive strains than when kept in strain-specific groups (Tindell and Craig, 1959; Biswas and Craig, 1970). Craig and his colleagues addressed this situation by selecting for part-year egg mass where sire families were intermingled or maintained separately in flocks (Quadeer et al., 1977a, 1977b). Interactions of sire families x social environment were generally unimportant for production traits except early egg weight. Heritability estimates from paternal half-sib correlations were lower in family-separated than intermingled flocks for all traits except egg weight.

When Darden and Marks (1988b) compared lines of Japanese quail bidirectionally selected for 4-week body weight under cafeteria or single-source diets in both environments, interactions were present for feed consumption (Table 35.1). The pattern of response differed depending on the direction of selection (high or low) and the environment in which selection occurred. Although in all cases consumption was greatest in the single-source environment, the largest difference between groups was for the high and low weight cafeteria-selected lines in the cafeteria environment. Dove (1935) observed considerable individual variation in ability of chickens to select adequate diets. Perhaps selection in the cafeteria environment finetuned the ability of quail to select a diet which optimizes (not necessarily maximizes) their growth potential.

Another example of genotype x environment interactions from earlier in this chapter is shown in Figure 35.3. Differences in taste preferences among parental and F_1 cross populations were dependent on the concentration of the test solutions. Moreover, inferences concerning genetic variation for discrimination varied with the concentration of the solution.

There is considerable evidence that genotype x environment interactions exist for various behaviors. A strain of White Leghorns exhibited similar amounts of prelaying pacing in light and dark pens, while a strain of Rhode Island Red and Light Sussex origin paced half as much in dark as in light pens (Wood-Gush, 1972). Comparing lines considered docile and flighty to humans, line x type of food interactions were observed for

Table 35.1. Mean feed consumption (g/bird) from 3 to 4 weeks of age by selection line (H = high weight, L = low weight, C = cafeteria feeding, S = single-source feeding) under both feeding environments. (Adapted from Darden and Marks, 1988b).

Feeding Environment	Selection Line			
	HC	HS	LC	LS
C	120	112	64	84
S	130	141	104	105

latency of time to eat (Murphy, 1977).

Returning to selected populations discussed earlier in this chapter, effects of rearing environment on mating frequency were measured in chickens selected for high and low mating frequency (Dunnington and Siegel, 1983). The lines were reared with sexes intermingled to eight weeks of age and then transferred to sex-separated flocks until about 28 weeks when mating trials were conducted. A valid question was whether the lines would respond similarly if reared with females to testing age as they did under the husbandry in which selection was practiced. Mating frequency was compared when males were reared with females through maturity vs. placing them in all-male flocks at six weeks. As seen in Table 35.2, genotype x rearing environment interactions were present. Rearing males in heterosexual flocks increased the frequency of maters in the unselected and low lines by approximately 20 percent, while essentially all males in the high line mated regardless of how they were raised.

Using lines selected for high and low plasma corticosterone response to social strife described earlier (Gross and Siegel, 1985) and lines bidirectionally selected for antibody response to sheep erythrocytes (Siegel and Gross, 1980), several experiments were conducted (eg. Gross, 1976; Gross and Siegel, 1981, 1982; Gross et al., 1984) to assess susceptibility to bacterial and viral infections with different levels of social strife. For Marek's disease, percentage of mortality and lesions were greater in the high than low corticosterone line and in an environment with higher than lower social strife (Table 35.3). Those chickens in the high corticosterone line and with higher social strife were ten times more susceptible than low corticosterone line chickens with low social strife. Comparisons involving Northern feather mite infestation mirrored those for Marek's disease. These results and those from other experiments imply that a social environment that may be optimum for one population may not be optimum for another and that drastic changes in husbandry may require time for populations to adapt.

At the commercial level, Hansen (1976) in a fascinating paper discussed differences among White Leghorn strains in their propensity for abnormal nervousness and hysteria in high density environments. Consistent with these observations, differences in agonistic behavior and vocalization patterns between White Leghorn strains housed in the same high density unit have been shown (Stone et al., 1984). To know the environmental history in which these strains were selected could contribute not only to our understanding of behavioral genetics of poultry, but to all of behavioral genetics.

Table 35.2. Percentages of males that mated, in lines of chickens selected for high and low mating frequency and the unselected control line, when reared in all-male and in sex-intermingled flocks. (Adapted from Cook and Siegel, 1974).

	Social environment	
Line	All-male	Sex-intermingled
High	100	95
Control	69	91
Low	43	65

Table 35.3. Percentage of mortality and lesions in response to Marek's disease exposure, and number of Northern feather mites, for lines of chickens selected for high and low plasma corticosterone response to social strife maintained in environments with higher and lower social strife. (Adapted from Gross, 1972, and Hall and Gross, 1975).

		Social Strife		
	Line	Higher	Lower	Pooled
Marek's	High	71	40	56
	Low	36	7	22
	Pooled	54	24	
Mites	High	57	132	94
	Low	409	2200	1304
	Pooled	233	1166	

CONCLUDING REMARKS

This chapter discusses influences of behaviors on gene pools of subsequent generations and modes of inheritance for an array of behaviors. The history of domestication provides numerous examples of the role of behaviors in poultry breeding. Experiments demonstrate that behaviors can be modified through selection and that genotype x environment interactions are common for many behaviors.

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Chapter 36. GENOTYPE x ENVIRONMENT INTERACTIONS

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The term 'genotype x environment interaction' is most commonly used to describe situations where different genotypes (for instance breeds, lines, strains) respond differently to different environments. These differences in genotype response not only include changes in mean performance but also include variability in performance of different genotypes. Another type of genotype x environment (G x E) interaction can occur in the comparison between the performance of a breeder's purebred selected lines and that of their crossbred progeny which are distributed to poultry producers. This comparison can involve the purebred parental and crossbred progeny populations being tested under either the same or different environmental conditions.

The potential importance of G x E interactions to both the poultry breeder and the poultry producer appears to have been first recognized by Munro (1936). He also noted that each individual phenotype is the result of an interaction between a specific genotype and a particular environment. These days, however, the term genotype x environment interaction is normally only used to refer to interactions occurring between, rather than within individuals.

Hull and Gowe (1962) observed that important G x E interactions tend to occur when the genotypes being compared differ substantially in combination with large environmental differences. Genotypic differences are expected to increase with increasing divergence between the environments under which the parent lines were selected. However, G x E interactions can occur among unselected lines.

The occurrence of G x E interactions can sometimes bias experimental comparisons if the experimental design is defective. Depending upon the comparison, interactions between genotype(s) and hatch, rearing environment, or vaccine type, to name but a few, can produce misleading information.

CLASSIFYING G x E INTERACTIONS

Various criteria have been proposed for categorizing G x E interactions. Haldane (1946) suggested four general types of interactions involving two genotypes and two environments; they are presented diagrammatically in Figure 36.1. Haldane's type 1 only represents a form of G x E interaction when one genotype is more variable in performance and/or more sensitive to environmental changes than the other genotype.

McBride (1958) suggested another approach, with G x E interactions being classified according to whether a major or minor environmental difference is involved, and whether the interaction is occurring between different genotypes from within the same population or between different populations. His four categories therefore were micro-environment intrapopulation, macro-environment intrapopulation, micro-environment interpopulation, and macro-environment interpopulation. Each of these four categories can be further subdivided according to Haldane's criteria.

In contrast to this approach, Falconer (1952) suggested that when a particular trait is being assessed in two different environments, it should be considered as being two distinct

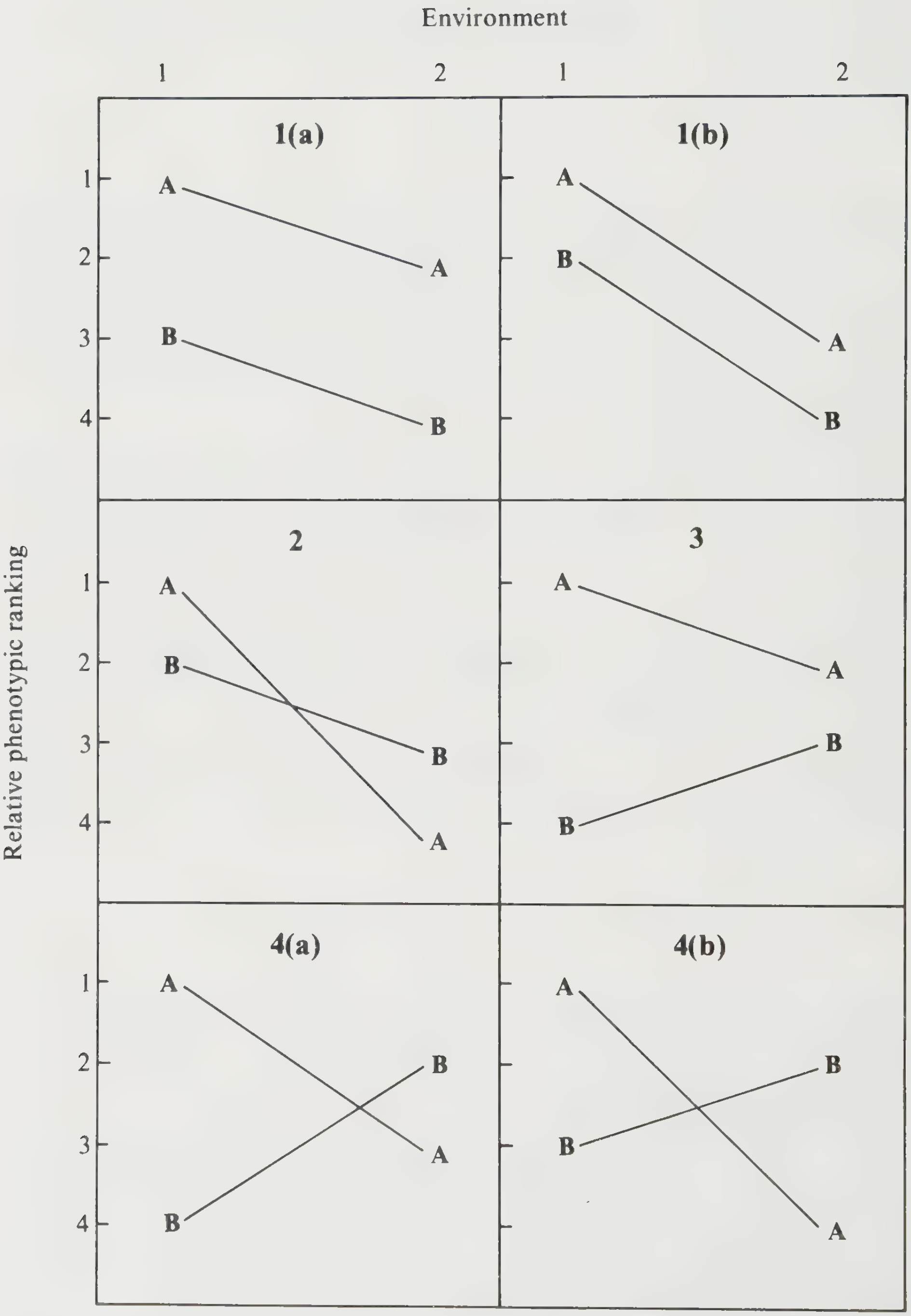

Figure 36.1. Types of G x E interactions suggested by Haldane (1946) for two genotypes, A and B.

but correlated traits. Using this approach, direct and correlated selection responses can be predicted from the genetic correlation between these two 'traits' in combination with their heritabilities and phenotypic variances.

Yamada (1970) suggested that experimental investigations into G x E interactions can be classified according to whether a static or a dynamic approach was used. The static approach involves comparing the performance of samples of existing populations tested in different environments. The dynamic approach involves the comparison of responses in lines derived from similar base populations and selected for the same traits while being exposed to different environments.

THE BREEDERS' PERSPECTIVE

Poultry breeders are not concerned with academic classifications of genotype x environment interactions. Their aim is to provide a product package of layer and/or broiler strains, with appropriate husbandry and management recommendations and technical support, that will maximize their return on capital invested by attracting an appropriate level of sales at the right price in the market(s) being serviced. A small breeder servicing a restricted geographic region will be less concerned with the possibility of G x E interactions than will a major poultry breeding organization distributing stock world-wide.

Poultry producers tend to choose a strain of birds from those available that, under their husbandry, management, and market conditions, will in their perception maximize return on capital invested. Factors such as employing an experienced hatchery manager proficient at consistently hatching healthy sturdy chickens can have a substantial impact on sales. However, it is mainly economic factors that finally determine the geographic distribution of poultry strains available for sale and the particular strain(s) that individual poultry producers purchase.

Over the years, the world's poultry industries, particularly in developed countries, have been characterized by a trend of improved control over the bird's environment. It is technically feasible to provide intensively housed poultry with an identical environment, irrespective as to whether they are located in Norway in winter or New Guinea in summer. However, with the present capital and operating costs of refrigerant air conditioning, it is not economically viable to provide such a sophisticated level of climate control. Therefore, poultry tend to be kept under environmental conditions (temperature, humidity, housing, stocking density, etc.) that vary widely from temperate to tropical climates and from country to country.

The environmental factors of most concern to the breeder are ambient temperature, humidity, and market factors. Ambient temperature can usually be modified to fall within acceptable limits by housing the birds in insulated sheds, by reducing the ventilation rate in cold climates, and by using evaporative cooling and fans in hot climates. However, poultry are still subjected to a broad range of temperatures. Market factors are generally beyond the control of the poultry breeder.

Present approach adopted by breeders. With the world-wide distribution of stocks, the major poultry breeders are confronted with the problem of determining the best strategy for satisfying a diversified customer range differing in market requirements and climatic conditions. The approach generally used has been to attempt to provide a selection environment intended to minimize the development of adverse interactions of potential economic importance. Breeders also monitor their commercial strains for the early detection of G x E interactions. If possible, any such interactions are removed or reduced in effect by the recommendation of cost-effective husbandry and/or management conditions that will provide an appropriate or nearly appropriate environment. An example of this approach includes strain-specific elevated nutrient requirements that can be simply and inexpensively satisfied by the addition to the diet of increased levels of the appropriate nutrient.

Occasionally, potentially important G x E interactions that cannot be eliminated or reduced by the cost-effective provision of specific environmental conditions are identified. In such cases, consideration is given to the economic viability of developing a specific strain or strains for that particular environment. Decisions on whether and when to develop special strain(s) depend on several factors including: the size of the market associated with that environment and its current and potential economic value to breeders, the relative profitabilities to breeders of the strains currently available to poultry producers operating in that environment, whether any specialized strain(s) have been developed for or in that environment and their profitability relative to the other strains available, the relative competitiveness and market share enjoyed by the breeder's own strain(s) in that environment, and the cost of providing a specialized strain for the environment and the increased market share likely to be captured by it.

Obviously it is economic factors that determine whether or not a breeder should develop a specialized strain to cater for particular environmental conditions. Examples where it has become financially viable to develop specialized lines for particular environments include: the replacement of dual purpose breeds with specialized meat and egg producing strains, the development of strains of layers producing brown-shelled eggs, the development of strains of layers with different mean egg weights since the return to egg producers for the various egg weight grades can differ substantially between markets, the development of red-feathered strains of broilers in addition to the white-feathered strains, and the development of broiler strains with either white or yellow skin.

Factors of concern to breeders. Bell (1970) provided an excellent summary of the practical questions confronting the poultry breeder regarding G x E interactions. These questions, which still remain unanswered today, are as follows. Will the importance of G x E interactions increase with the world-wide distribution of poultry strains? Should the selection lines be bred under optimal or stress environments? Should the breeding goal be general or specialized adaptability?

In considering these questions, Bell (1970) suggested that there were four not necessarily mutually exclusive alternatives: to select under a uniform and optimum environment, to select under environmental conditions other than optimum, to develop specialized strains or genotypes uniquely adapted to specific environmental conditions, and to select for general adaptability to a wide range of environments. The reader is referred to Bell (1970) for a thorough discussion on these alternatives. Briefly, all of them have been utilized, depending upon the economic importance and nature of the particular G x E interaction.

The first alternative, to select under a uniform and optimum environment, has been and is largely being followed with respect to exposure to disease organisms. Most nucleus poultry selection lines are held under isolated quarantine conditions in conjunction with disease eradication/limitation programs, which are atypical of practical commercial environments with their higher incidences and greater risk of exposure to pathogens. However, a few breeding companies consider that exposure to pathogens assists in selection for viability 'in the field' and are less strict regarding disease preventative measures. Also, from the husbandry point of view, nucleus layer selection lines are normally evaluated in single-bird cages whereas their commercial daughters are usually housed in multiple-bird cages. The second alternative, to select under environmental conditions that are not optimum, is practiced in that as far as is strategically and economically sensible, the husbandry and management conditions of the nucleus selection lines are as similar as possible to the average commercial conditions under which their progeny are expected to perform. Thus, for example, the diet fed and the standard of climate control (for instance lighting intensity, daylength, ambient temperature) closely approximate current commercial practice. Examples of the third alternative, to develop strains adapted to specific environments, include the development of specialized meat and egg producing strains, layer strains with different egg shell colors, and broiler strains with either red or white feathers. Most major poultry breeders supplying commercial stock to producers in different countries test their commercial strains at a number of climatically and geographically different locations under typical local commercial conditions. This approach can be an example of the fourth alternative, selection for general adaptability, if the information obtained is being used to select for general adaptability either between different strain-cross combinations or when selecting between sire families within the nucleus selection lines.

In addition, the commercial strain is normally a crossbred, usually involving between two and four parental lines. One of the reasons for marketing a crossbred is its greater tolerance and resistance to environmental stress, thus further reducing the adverse impact of G x E interactions. Conversely, under extremely favorable environmental conditions a purebred line may outperform a 'buffered' crossbred line due to beneficial G x E interactions.

EXAMPLES OF G x E INTERACTIONS

In considering G x E interactions, the environment is defined in the broadest sense and includes economic and market conditions in addition to the more usually considered factors such as husbandry, management, type of housing, climate, and geographic location. Table 36.1 represents an attempt to specify various environmental factors that can differ between poultry farms. Many of these factors can also differ between groups of poultry located at the same farm.

An unusual example of a G x E interaction involving broiler breeders was due to cockerels from one of the sire lines having pea combs (Herbert, 1988, personal communication). The restricted-feeding treatment involved cockerels and pullets being fed separate diets, with cockerels being excluded from the pullet diet by the size of the feeder openings. However, cockerels with pea combs were able to feed through these openings. This resulted in the cockerels being fully fed, and thus heavier and with an increased incidence of leg problems, and the pullets being more severely restricted.

Neighboring egg producers with identical husbandry and management systems can have completely different poultry stock requirements. One producer could be directly supplying the public and receiving a premium for large brown-shelled eggs. The other producer could be consigning all eggs to the local egg marketing authority and being paid according to the total mass of saleable eggs without regard to either the distribution of egg weight grades or shell color.

Although G x E interactions can involve many of the environmental factors listed in Table 36.1, the ensuing discussion has largely been limited to a consideration of those environmental factors that are not always under the cost-effective control of the poultry producer and/or poultry breeder. These factors are ambient temperature, humidity, geographic location and specific market requirements. Humidity is confounded with

Table 36.1. Types of environmental factors that can differ between poultry farms.

Husbandry/management
- nutrition
  - ingredients used
  - diet specifications
  - standard of formulation
  - *ad lib.* or restricted feeding
- water quality
- single or multiple age flocks (all-in all-out, or continuous replacement)
- age of birds at disposal
- standard of husbandry/management provided
- disease status and preventative measures
- vaccine type
- disease monitoring and control programs
- manual or automatic egg collection
- standard of climate control
  - lighting (daylength, light intensity)
  - temperature
  - humidity
  - air speed
  - air quality (filtered or unfiltered)

Economic factors
- capital costs
  - land cost/value
  - building costs (building codes, materials, labor)
  - cost of long-term finance
- operating costs
  - labor
  - taxation rates
  - effluent/waste disposal
  - feed
  - transport
  - replacement birds
  - disposal of unwanted stock
  - cost of short-term finance
  - disease preventative and control measures
- opportunity costs (capital invested, alternative land uses)
- market factors
  - size of market
  - supply/demand factors
  - product categories and value (egg weight grades, broiler weight grades)
- cost of complying with local animal welfare requirements

Housing
- single-bird cages
- multiple-bird cages
- deep litter
- open range
- type of equipment (feeders, waterers, etc.)

Geographic location
- climatic factors (diurnal and seasonal variation)
  - daylength
  - light intensity
  - temperature
  - humidity
  - air speed
  - air quality
  - air pressure
- endemic diseases
- proximity to other poultry flocks
- urban planning requirements
  - buildings
  - land use
  - waste disposal
- labor
  - availability
  - level of expertise
- proximity to markets
- availability of services
  - electricity
  - water
  - gas
  - technical services (service personnel, veterinarians, advisors, etc.)
- proximity to suppliers
  - feed mill or feed ingredients
  - replacement stock

Other factors
- social environment (schools, clubs, churches, etc.)

ambient temperature and geographic location, and thus it cannot be considered separately.

Interactions between genotype and nutrition have been reviewed by Hutt (1961) and Quisenberry (1969). Genotype x disease interactions occur when genotypes differing in genetic resistance to a particular pathogen are compared both in the presence and absence of that pathogen. Gavora and Spencer (1978) have reviewed various poultry diseases to which there is at least some degree of genetic resistance. Detailed review of these topics is also given in Chapters 33 and 34.

Market requirements. Generally, the manipulation of market requirements to approximate the specifications of a particular poultry strain is beyond the scope of the breeder. He/she is mainly concerned with the market factors determining the return to the poultry producer since these are not necessarily identical to those that determine the return to the retailer. In some markets, for example, the wholesaler purchases eggs on a total weight basis and then prices the various egg weight grades to the consumer according to the prevailing market conditions of supply and demand. This is the ideal situation which, if universally adopted, would enable the breeder to select simply for total quantity of eggs produced irrespective of the egg weight distribution or egg shell color. However, in many markets, the prevailing consumer preferences, in combination with supply and demand for the various egg weight categories, determine the return to the egg producer. Various market requirements for egg weight categories are discussed in Chapter 40.

As also discussed in Chapter 40, broiler breeders have a diverse range of markets to satisfy. The whole carcass market consists of a range of weight categories varying from 1-3 kg. The cut-up market requires very uniform birds and has two components, retail and fast food. The retail market usually requires a larger carcass. There is also the reconstituted/processed meat market which includes chicken nuggets, fingers, loaves, sausages, and hamburgers. Markets can also differ in consumer preference for yellow or white skin color and for red or white feather color. Both of these traits are manipulated by the infusion of a major gene into the parental lines. With the exception of a few of the small specialized markets, for instance for gourmet chickens involving slow growing birds processed at an older age, most broiler producers have satisfied these markets by manipulating the slaughter age. However, some broiler breeding organizations are currently developing specialized lines, one for satisfying the smaller carcass market and the other for the larger carcass market. Since the smaller carcass line is slaughtered before leg abnormalities become a serious problem, greater selection pressure can be placed on the other traits of interest. Thus, within these general carcass weight categories, broiler producers desire a rapidly growing chicken with good conformation and feed conversion, a low variability in body weights, and a high yield of saleable meat, especially of prime cuts such as breast meat.

The only ways in which a breeder can attempt to influence market requirements is by promoting as desirable the specific features of his strain(s) and/or lobbying with the poultry wholesalers who in some countries are statutory authorities. Thus, in most situations, it is beyond the breeder's control to manipulate the market requirements to suit his own stock.

Temperature. The effect of extremes of temperature on the relative performance and survival of different strains/breeds of poultry has been the subject of many studies. Hutt (1938) reported that the number of deaths from heat stress during a heat wave differed significantly between White Leghorns (1.8 percent), Rhode Island Reds (5.3 percent), and Barred Plymouth Rocks (5.2 percent). He observed that tolerance to extreme heat appeared to be unrelated to either body size or rate of lay. He also noted that poultry introduced into tropical countries from Europe or North America were notoriously less tolerant of the climatic conditions than the indigenous stock.

Using a temperature-controlled room, Lee et al. (1945) studied the effects of high ambient temperature (41°C) on White Leghorns, Brown Leghorns, White Wyandottes, Australorps, and Rhode Island Reds. They observed that, in general, Australorps and Rhode Island Reds were most susceptible and White Leghorns least susceptible to heat stress. Fox (1951) suggested that the better tolerance of White Leghorns to heat stress was associated with a behavioral trait, namely their greater persistency to consume water under hot conditions. He observed that when White Leghorn hens were exposed to 42°C without drinking water, on average they died sooner than White Plymouth Rock hens (89 vs. 112 min). However, when drinking water was provided, White Leghorn hens tended to survive longer than Rhode Island Red hens (281 vs. 150 min). The survival time of hens was apparently not influenced by their breed, being simply related to an individual's persistency in continuing to consume water. In one experiment a White Leghorn hen and two New Hampshire hens that drank continuously survived a heat exposure period of 11.5 h in good condition.

Kheireldin and Shaffner (1957) exposed day-old New Hampshire chicks to temperatures up to 43°C and then observed their growth rate. They concluded from the large variation in mortalities between sire and dam families that heat tolerance is controlled genetically.

Huston and Joiner (1957) compared the laying performances of New Hampshire, White Plymouth Rock, and White Leghorn pullets when housed in individual cages under two temperature regimes. One group had been maintained from hatching at a constant 32°C while the other group had been provided with normal brooding temperatures before being exposed to the naturally occurring and variable ambient temperature. The White Leghorns performed similarly irrespective of the temperature regime, whereas the rates of lay for the other two breeds were reduced in the high temperature environment.

Clark and Amin (1965) compared the laying performance of five strains of White Leghorns that had been bred at different locations ranging from relatively cool to warm and with a mean difference of 14°C. The hens were housed in individual cages in two temperature-controlled experimental rooms and in a conventional 'uncontrolled' poultry house. One experimental room was maintained at 21°C during the day and 38°C at night, while the other room was maintained continuously at 18°C. Differences between strains in egg production tended to reflect the environment under which they had been bred. Although most of the other traits studied were affected by the temperature differences, they did not show any genotype x environment interactions.

Wilson et al. (1966) found that a strain of White Leghorns exposed to 41°C and 75 percent humidity when four weeks old differed significantly between sire and dam families in survival time. They estimated the heritability for survival time to be 0.45 ± 0.15 and predicted that selection for either heat tolerance or susceptibility at this age would be successful. This population was then divergently selected for heat tolerance/susceptibility at five weeks of age. Wilson et al. (1975) reported that, after four generations of progeny selection, the mean survival time of these two lines was 56 and 86 minutes. They observed that a chicken's body temperature after 30 min of heat stress was negatively correlated with survival time. The more heat tolerant chickens tended to have a lighter body weight and showed a greater weight loss during heat stress. At least some of this greater weight loss would have been due to the longer survival time. Bohren et al. (1982b) reared samples of these lines from five to nine weeks of age at either 21 or 32°C and then subjected them to 41°C. Chickens from the heat resistant line were slower growing with a lower mortality level during heat stress. Irrespective of their selection history, birds reared in the hot environment were lighter at nine weeks of age and also more likely to survive the heat stress than birds reared in the cold environment. The environmental correlation between body weight and heat stress susceptibility was estimated to be 0.75.

Washburn et al. (1980) investigated the response to heat stress (50°C) at eight weeks of age for up to four h in three lines differing in body weight. These lines were a control flock, a line under selection for increased growth rate, and a broiler line in which growth rate selection had been relaxed. Samples of each of these three lines were reared to eight weeks of age under either *ad libitum* or restricted feeding. Resistance to heat stress was greater in smaller chickens irrespective as to whether their reduced body size was due to feed restriction during rearing and/or to their selection history.

Bohren et al. (1981) selected two replicates of White Leghorn chickens for four generations for increased or decreased five- to nine-week growth rate in either a hot (32°C) or a cold (21°C) environment. In each generation, progeny from the selection lines and the control lines were compared under both environments. All lines grew faster in the cooler environment. There was no evidence of any interaction between direction of selection or the selection environment for growth rate under either temperature. The survival time of these lines to a temperature stress (41°C) when nine weeks old, after being raised in either a hot or a cold environment, was investigated (Bohren et al., 1982a). In some of the experiments, a period of acclimatization at 38°C was provided prior to raising the temperature to 41°C. Increased survival time was associated with being raised in a hot environment and being provided with a period of acclimatization, there being no influence of selection history.

Mukherjee et al. (1980) compared the performance of a crossbred brown egg laying strain from hatching to 68 weeks of age in both a cool West German and a warm Malaysian environment. The experimental pullets were obtained from assortative matings between light, medium, or heavy cockerels and pullets selected on the basis of the mature body weight of their daughters. Statistically significant sire family x temperature (geographic location) interactions were reported for age at sexual maturity, egg production, egg weight, food consumption, and total mass of eggs produced. However, these interaction effects were relatively minor in comparison to differences between sire families and locations. Rate of egg production was negatively correlated with 40-week body weight in Malaysia (r=-0.4), while in West Germany there was a low positive correlation between these two traits (r=0.1). In a further experiment in this series, Alihussain-Gadhia et al. (1984) found adult body weight to be an important factor underlying the sire family x location interactions observed for various egg production traits. The optimum 40-week body weight for maximizing productivity was lower in Malaysia (1.9 kg) than in West Germany (2.2 kg). Horst (1985) presented additional information from this investigation that further supported the observation that within breeds/strains, smaller birds appear to be better adapted to withstand heat stress. None of these reports presented any information on individual water consumption. Thus a possible relationship between water consumption and either body weight or egg production rate under tropical conditions cannot be explored.

Marks and Huston (1973) observed that Japanese quail selected for increased four-week body weight were less tolerant of heat stress at 45°C than an unselected control line. Chahil et al. (1975) selected four replicate lines of Japanese quail for increased three- to five-week growth rate at either 37 or 21°C. Progeny from the third selected generation were tested under both temperature regimes. Although birds from both selection environments grew faster and at a similar rate in the cooler environment, the reduction in growth rate in the high temperature environment was significantly less for the lines selected in that environment.

With the exception of one Japanese quail selection experiment, all of the available experimental evidence indicates that genotype x ambient temperature interactions are of little consequence for broiler chickens. However in the U.S.A., differences in growth rates between commercial broiler strains can be substantially reduced or even disappear when they are reared under extremely hot conditions (Herbert, 1988, personal communication). There is considerable evidence for layer strains that genotype x environment interactions are present for tolerance to high and low temperatures, with hens generally performing best in the environment in which they were bred.

Geographic location and/or housing — layers. Much of the early work into genotype x location or genotype x housing systems was carried out by scientists associated with the Canada Department of Agriculture (Gutteridge and O'Neil, 1942b; Gowe and Wakely, 1954; Gowe, 1956; Proudfoot et al., 1957; Hull and Gowe, 1962; Gowe et al., 1962; Hull et al., 1963). Except for the first investigation, which used Barred Plymouth Rocks, all of these comparisons utilized White Leghorns. Also with the possible exception of the first experiment, all management and husbandry practices at the various locations were standardized as closely as possible. In the experiments that involved a number of locations, the few statistically significant genotype x location interactions were too small to be of any practical importance. The one experiment that compared the performance of different strains of layers housed in either single cages or intermingled in pens found statistically highly significant genotype x housing interactions for survivor egg production and mature body weight. As discussed later, these interactions could have been due to social competition between the different strains of hens when housed in pens. Another experiment compared three strains of layers when housed in pens either separately or intermingled. As discussed later, the absence of any statistically significant strain x housing treatment interaction could have been due to the comparison of strains with similar levels of social competition or aggression. Some of the experiments included the rearing treatments of restricted vs. *ad libitum* feeding. It was concluded that to avoid possible feeding treatment x genotype interactions, where laying stock was to be reared under restricted feeding to delay sexual maturity and to increase initial egg size, the parent stock should be selected under this feeding regime, a practice universally followed now.

In contrast to the Canadian group, scientists at Iowa State University (Hill and Nordskog, 1956; Nordskog and Kempthorne, 1960) obtained experimental evidence for important and statistically highly significant interactions between genotypes and locations for age at sexual maturity, egg production, egg weight, and adult mortality. In agreement with the Canadian group, they found statistically significant interactions between genotypes and type of housing (cages vs. pens) for egg production. These comparisons also differed from the Canadian experiments in that no attempt was made to standardize husbandry and management conditions between different locations. In addition, hatch effects were confounded with location effects which could also have contributed to the genotype x location interactions observed.

Other scientists investigating genotype x location interactions (Abplanalp and Marrou, 1961; Tindell et al., 1967b; Marks et al., 1969) found either no consistent interactions or no statistically significant interactions of practical importance.

McBride (1964) and Craig and Toth (1969) reviewed the experimental evidence and also investigated the relationship between social dominance or aggression and laying performance when layer strains are housed in pens. Within any one pen, the more aggressive hens tended to have superior production, as indicated by larger eggs and a greater rate of lay. However, the most productive pens tended to contain less aggressive groups of layers. Thus selection for increased egg production among hens housed in pens could confound laying performance with aggression. On the other hand, selection among hens housed in single cages is unlikely to be influenced by the level of aggression. Genotype x housing interactions are therefore most likely to occur when strains differing substantially in their levels of aggression are intermingled in the same pen or multiple-bird cage. On this basis, genotype x housing interactions would be expected to be absent or of a low order when layer strains selected under single-bird cage conditions are separately housed in groups.

Other investigators (Hale, 1961; Francis and Roberson, 1963; Logan, 1965; Christmas et al., 1972) compared the performance of layers in cages and in pens or under semi-intensive conditions. They found either no evidence or no consistent evidence for the presence of genotype x housing interactions. These results are in contrast to those of the Canadian and Iowa groups and could be due to the strains used in these later experiments having been selected for increased egg production when housed in single-bird cages.

Most of the comparisons involving layer strains housed in cages in various group sizes and stocking densities failed to detect any statistically significant genotype x housing interactions of practical importance (Logan, 1965; Adams and Jackson, 1970; Marks et al., 1970; Connor and Burton, 1975). An exception was Aitken et al. (1973) who compared seven White Leghorn layer strains when housed either singly or in pairs in small (20 x 41 cm) cages. They observed highly significant genotype x housing interaction effects for egg production, egg weight, body weight, and feed consumption. However, despite the hens not having been beak-trimmed, this interaction was apparently due to space restriction rather than to social competition effects. The strains with the largest adult body weights also tended to show the greatest reduction in all of these traits when housed in pairs.

When Bell et al. (1983) compared dwarf and normal layer strains which were otherwise identical, the only significant strain x housing interaction was for feed efficiency. Each strain was housed separately at either two or three hens per cage. The interaction was due to dwarf hens having a superior feed efficiency when housed three per cage, with the reverse being the case for normal-sized hens.

In one experiment (Lowry and Abplanalp, 1970) selection was conducted for seven generations for increased egg production in White Leghorns housed either in pens or in single-bird cages. The three paired comparisons had each been derived from different but related populations. Hens from different replicates were intermingled in the pens or randomly allocated to the same blocks of cages. Thus hens from each line were in close social contact and in direct competition in the pens with hens from the other lines. Samples of all six selection lines were compared at Generations 6 and 7 either intermingled in pens or randomly allocated to single cages. The results indicated that: on average selection in the cage environment produced a greater response than did selection in the pen environment; each selection line performed best when tested in its selection environment; in two of the three paired comparisons, cage-selected hens were superior to pen-selected hens when tested in pens; in all comparisons, cage-selected hens were superior to pen-selected hens when tested in cages. Since the intermingling of strains in the pens could have favored the pen-selected lines, the cage-selected lines may have performed even better in pens had they been housed separately.

Horn (1982a) reviewed the scientific literature relating to genotype x housing and/or location interactions in layers. He noted that the majority of comparisons had involved Leghorn hens, and concluded that although the experimental evidence tended to be somewhat contradictory, genotype x housing interactions were likely to be of minor consequence to poultry breeders.

There has been a steady trend toward economic viability of modifying the hen's environment to that specified by the breeder for maximizing production. This trend is expected to continue. As discussed elsewhere, the main environmental factors considered responsible for economically important genotype x location interactions are major differences in ambient temperature/humidity and different market requirements reflected in the return to the producer. However, the introduction of legislative restrictions relating to the housing of layers in cages, at the behest of animal welfare lobbyists, could make it economically necessary to reduce the precision of control over the hen's environment. Such legislation would encourage poultry breeders to shift their breeding operations to countries unlikely to ban laying cages. This is because selection for increased egg production between individually caged hens appears to be the most efficient, effective, and economical method of improving the egg production of their crossbred progeny whether housed in cages or on the floor. However, breeding companies could afford to individually house their nucleus selection line hens in laying cages too large for the cost-effective production of eggs by commercial farmers. Any requirements that laying hens be housed on deep litter and/or in open yards reduce the farmer's control over the external environment. This, in turn, could lead to the occurrence of some economically important genotype x environment interactions.

Geographic location — broilers. Tindell et al. (1967a) provided an excellent review of the early work while Horn (1982a) summarized the more recent investigations into the possible importance of genotype x location interactions in broiler chickens. These various experiments have either found genotype x location interactions to be absent or to account for less than three percent of the phenotypic variation in body weight at processing age (Gutteridge and O'Neil, 1942a; Merritt and Gowe, 1956; Lewis and Blow, 1965; Tindell et al., 1967a, 1968; Marks et al., 1972; Horn, 1982a). An interim report (Cook et al., 1962), based on a preliminary analysis of the results of the comparison reported by Tindell et al. (1967a) had suggested that genotype x location interactions could be of practical importance. Most of these investigations compared various broiler strains when intermingled together and, more importantly, did not attempt to standardize diets or husbandry and management procedures across locations.

Horn (1982a) suggested that the small contribution of genotype x environment interactions to the total variance in broiler chicken body weights was possibly due to the following reasons: there has been a substantial reduction in the number of primary breeders leading to a loss in genetic variability; there has been an exchange of genetic material between breeders; most primary breeders have used similar selection procedures under similar environmental conditions; the various male and female broiler strains possibly originate from a small number of base populations.

The current practice of standardizing the husbandry, management, and dietary conditions according to the breeder's recommendations should even further reduce the minor contribution of G x E interactions to variations among broiler strains. However, due to the extremely large numbers of broiler chickens being raised, even very small interactions can be economically important.

PUREBRED PARENTAL LINES AND CROSSBRED PERFORMANCE

As discussed in Chapter 40, most poultry breeding organizations maintain their purebred nucleus selection lines in a closely controlled test farm environment, while aiming to produce terminal crossbreds that will perform well over a range of commercial farm environments. The test farm is usually quarantined from outside stock, being highly atypical of the commercial farm environment with its higher incidence of pathogens and greater risk of exposure to them. Nucleus layer selection lines are normally evaluated in single-bird cages whereas their crossbred progeny are usually housed in multiple-bird cages.

Table 36.2 indicates the relationship existing between purebred parental and terminal crossbred progeny lines and between test farm and commercial farm environments. The traditional G x E interaction is concerned with quadrants A and B for purebred lines and quadrants C and D for crossbred lines. The relationship between purebred selection and crossbred response, a form of G x E interaction involving changing genotypes, is concerned with quadrants A and C at the test farm level, and quadrants B and D at the commercial farm level. Poultry breeders are concerned with the relationship between quadrants A and D which involves a combination of differing genotypes and different environments. The commercial farm environment can cover a wide range of geographic, husbandry, and market conditions.

Various workers (Biswas and Craig, 1969; Horn, 1982b; Pirchner, 1976; Pirchner and von Krosigk, 1973; and others) have investigated the genetic relationship between purebred and crossbred performance for some egg production and body weight traits. In general, the genetic correlation tended to reflect the heritability of the trait, being high for egg weight, egg specific gravity, age at sexual maturity, and body weight, and intermediate for egg production.

It is well documented (Cole and Hutt, 1973; Ayyagari et al., 1982; Gowe and Fairfull, 1982; and others) that response to selection for egg production within purebred lines is also manifested in crosses between the lines. However, the performance of the crossbred hens can also be influenced by heterosis and reciprocal effects. Breeding organizations invariably use testcross information to choose appropriate strain crosses, not only to exploit heterosis and reciprocal effects, but also to capitalize on complementation and adaptation to various commercial farm conditions. Testcrosses are normally evaluated under typical commercial conditions. If the testcrosses are pedigreed to the sire family, crossbred information may also be used to assist in the selection process within the parental lines. A few poultry breeding organizations claim to have made extensive use of reciprocal recurrent selection in developing their strain cross layers. However, as discussed in detail in Chapters 37 and 40, most commercial layer strains are believed to have been developed using pureline selection.

Environment x heterosis interactions. The degree of heterosis shown by a particular cross can be influenced by the environment, since crossbred animals are expected to be both more uniform and less influenced by environmental factors than their purebred parental lines. This type of interaction is discussed in Chapter 37.

Table 36.2. Classification of G x E interactions according to the relationships between purebred parental and terminal crossbred progeny lines and between test farm and commercial farm environments.

	Environment	
Genotype	Test farm	Commercial farm
Purebred parental lines	A	B
Crossbred progeny lines	C	D

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Chapter 37. HETEROSIS

R.W. Fairfull

The poultry industry has a history of using breed crosses and, more recently, strain crosses in poultry production, mainly to take advantage of heterosis. As the primary (or grandparent) stocks must reproduce the parent stocks of the commercial bird, it is relatively easy to introduce crossing into the process. By using a breeding program based on crossing, the breeder can combine stocks that complement each other and, in meat stocks, can develop specialized sire and dam lines. In addition, by crossing to produce the commercial stock, the breeder can control the release of primary (pure) lines. Most commercial stocks are now three-way or four-way crosses, or occasionally, two-way crosses. Two effects, heterosis and reciprocal effects, are important in poultry crosses.

Heterosis is the deviation between the cross and midparent means. The phenomenon of heterosis (or more narrowly *hybrid vigor*) was recognized by the late 18th century (Zirkle pp. 1-13 in Gowen, 1952). Two basic genetic mechanisms have been proposed to account for heterosis: *dominance* (interactions among alleles at the same locus) and *epistasis* (interactions between different loci).

The first theories of heterosis involved dominance (including overdominance) and were only proposed in the early 1900s (Bruce, 1910; Keeble and Pellew, 1910; Shull, 1911; Jones, 1917) with supporting evidence from crops (Richey and Sprague, 1931; Jones, 1945; Gustafsson, 1946, 1947). Heterosis caused by dominance is proportional to heterozygosity and, on average, is expected to be equal in two-, three-, and four-way crosses (heterozygous at all loci, in terms of strain or breed of origin). F_2s (heterozygous at 50 percent of loci) are expected to exhibit half the heterosis of two-way crosses. Thus, with known two-way cross performance, heterosis produced by dominance in subsequent crosses can be predicted (Dickerson, 1973) keeping in mind the limits of accuracy of prediction, and that changes in environment and other deviations may affect the estimates. Evidence supporting dominance as a cause of heterosis in animals is common for a wide array of traits (see Fairfull and Gowe, 1986) and dominance was broadly believed to be the only cause of heterosis.

However, recent work has shown clearly that epistasis is a major mechanism of heterosis in chicken stocks — in Australorp x Leghorn crosses (Sheridan and Randall, 1977; Sheridan, 1980), in Leghorn strain crosses (Fairfull et al., 1985, 1987) and in inbred lines from a common background (Haggar, 1986). Fairfull et al. (1985, 1987) showed that both dominance and epistasis were important in heterosis for egg production traits in Leghorn strain crosses.

Heterosis caused by epistasis can result from many genetic interactions (Hayman and Mather, 1955). However, as the chances for recombination increase, due to the use of more strains or generations, heterosis caused by epistatic interactions decreases through recombination of favorable gene combinations (*recombination loss*). Recombination gains (Haggar, 1986) are not unknown. In order to calculate expectations, the type of epistatic interaction must be known. Sheridan (1980) presents expectations for one such interaction, 'parental epistasis'. For the most part, heterosis resulting from epistasis is difficult or impossible to predict because the number and type of interactions are usually unknown and could also be affected by dominance.

There are three levels of heterosis: individual, maternal, and paternal (Dickerson, 1973). Unless specifically stated otherwise, heterosis in this chapter will mean individual heterosis. Most of the crossing work cited here incorporates only two-way crosses where only individual heterosis is possible.

Reciprocal effects or reciprocal differences are the deviations between the crosses of two parental strains or breeds in which their roles as male or female parents are reversed (i.e. 1x2 vs. 2x1). Reciprocal effects have been dismissed as unimportant at times and, outside of disease and disease resistance studies, are given little theoretical consideration. However, in practice, reciprocal effects are very important to meat and egg production in poultry. Breeders know that the economic merit of progeny resulting from crossing a male of strain (or breed) 1 with a female of strain 2 will likely differ greatly from those resulting from crossing a strain 2 male with a strain 1 female. Gowe and Fairfull (1982a) and Fairfull et al. (1983) have reported important reciprocal effects for most traits of commercial significance in white egg stocks of chickens.

Reciprocal effects are more of a consideration in layers than in meat birds. In meat birds, the lines used as male and female parents derive from different genetic backgrounds. Selection emphasis and the suite of traits selected usually differ in male and female lines. In male lines, growth, health (increasingly freedom from crippling), and male reproduction are the important traits, of which growth is the most emphasized. In female lines, less emphasis is placed on growth and more on female reproduction, especially on egg production. Thus, the reciprocal of a commercial meat cross is of no interest and has no logical practical value.

HETEROSIS OF PRODUCTION TRAITS

Chickens

Egg traits. Heterosis for egg production traits in poultry is consistent and substantial, but highly variable (-3 to 40 percent), commonly 10 percent or higher (Table 37.1). Epistasis results either in recombination loss, i.e. three- and four-way crosses have significantly and successively poorer performance than two-way crosses (Dickerson, 1965; Sheridan and Randall, 1977; Fairfull et al., 1985, 1987), or less commonly in recombination gain (Haggar, 1986) as has been shown for egg production traits. Heterosis for egg production in Leghorn crosses involves both dominance and epistasis (Fairfull et al., 1985, 1987) ranging from -3 to 30 percent (in two-way crosses) and is generally lower than the heterosis of Australorp x Leghorn crosses (15 to 40 percent) which seems to result entirely from epistasis (Sheridan and Randall, 1977; Sheridan, 1980).

In absolute terms heterosis for egg production does not decrease significantly with increasing parental performance (Fairfull, 1982; Fairfull et al., 1983), although percentage heterosis decreases with increasing parental performance. For example, the superiority of a cross in terms of number of eggs would be expected to be the same on average no matter how good or how poor the parental line performance. There may be very little or a great deal of heterosis expressed by a particular cross, but this cannot be predicted from parental performance alone. Thus, the crosses of the better parental strains can be expected to maintain the average superiority of their parents.

Fairfull et al. (1985, 1987) found maternal heterosis for early egg production significant in two traits — hen-housed production and rate of lay. Early maternal heterosis was large enough for rate of lay to influence the full-year rate significantly. Thus, a strain-cross dam will increase the total heterosis for early egg production. Maternal heterosis was

Table 37.1. Mean percent heterosis[1] of chickens (average plus range in parentheses where appropriate) for viability (VIAB), hen-housed egg production (HHP), hen-day rate of egg production (HDR), egg yield (EYLD), feed conversion (CONV), age at first egg (AFE), egg weight (EWT), egg specific gravity (SGR), Haugh units (HAU), percentage of eggs free of blood spots (BSP), and body weight (BWT), from the literature[2].

Reference	VIAB	HHP	HDR	EYLD	CONV	AFE	EWT	SGR	HAU	BSP	BWT
TWO-WAY CROSSES											
First egg production cycle											
Leghorn crosses	0(-9/5)	12(-3/30)	8(6/11)	16(10/25)	-5(-3/-6)	-4(-9/0)	2(0/4)	0	1(0/1)	0(-1/1)	3(0/5)^{M}
Rhode Island Red (RIR) crosses	2(0/5)	7(-1/11)					2(1/3)				3(0/6)^{M}
Australorp x Leghorn crosses	12(-1/24)	29(15/40)	18	25(21/31)	-11(-16/0)	-5(-3/-7)	4(3/5)				5^{M}
Leghorn x RIR crosses	-2	14(13/14)			9		2				0^{M}
Other egg-type crosses	-1(-2/0)	4(-3/15)	2(1/2)	6(5/6)	-4(-9/-1)	-4	0(-3/2)				0(-3/3)^{M}
Meat-type crosses	1(-5/5)	19(18/20)			11(7/16)	-4(-3/5)	3(2/3)				4(0/10)^{E}
Second egg production cycle											
Leghorn crosses	4(1/7)	15(13/16)	13(9/16)	16	-8		2	0	-1	-2	4(2/5)^{M}
RIR crosses	2(0/3)	10(8/11)					3				5(4/6)^{M}
THREE-WAY CROSSES											
Leghorn crosses	2	10	9	12	0	-3	2	0	0	-1	5^{M}
RIR crosses				-9(-18/-1)							-4(-12/4)^{I}
FOUR-WAY CROSSES											
Leghorn crosses	2	6	7	9	1	-2	2	0	0	-1	5^{M}
F_2 CROSSES											
Leghorn crosses	2	6	4	7	1	0	1	0	0	-1	3^{M}
Australorp x Leghorn crosses		-5	-3	0		-2	3				
F_3 CROSSES											
Australorp x Leghorn crosses		3	2	3		-2	1				

---

[1] Percentage of midparent value. [2] Al-Soudi and Al-Jebouri (1979), Damme et al. (1986), Fairfull et al. (1987), Jain and Chaudhry (1985), Orozco and Campo (1975), Sah et al. (1985) and for the remainder see Fairfull and Gowe (1986). ^{E} Early body weight in weeks 1 to 10, ^{I} intermediate body weight in weeks 11 to 20, ^{M} mature body weight.

insignificant for other egg production traits and for sexual maturity.

Heterosis for sexual maturity (AFE) ranges from 0 to -9 percent and closely approximates expectations resulting from dominance in three- and four-way crosses (i.e. equal to that of two-way crosses); however, heterosis in F_2s (Fairfull et al., 1985, 1987) or F_3s (Sheridan and Randall, 1977) often differs from such expectations and this appears to be the result of additive x additive epistasis (Fairfull et al., 1985, 1987). Heterosis for egg weight is somewhat lower (-3 to 5 percent) and conforms closely to expectations that would result from dominance alone (Fairfull et al., 1985, 1987). No linear relationship exists between heterosis and parental performance for egg weight (Fairfull et al., 1983). For egg quality traits (egg specific gravity, Haugh units, and percentage of eggs free of blood spots) there is little or no heterosis (see Table 37.1).

Body weight and growth traits. In meat stocks, early growth is vital. In broilers, heterosis for body weight is close to zero at one week of age and earlier, but increases to about 2-10 percent by 8-10 weeks of age (see Table 37.1). This heterosis is very important as broilers now reach market size in about 42 days or less. Adults of broiler stocks are usually reared under feed restriction regimes, and thus, although mature body size is important, heterosis estimates for body size in adults of breeding stocks would be misleading and of doubtful value (unless a negative deviation from the midparent can be found so as to alleviate maintenance requirements).

From hatch to ten weeks of age, Verma and Chaudhry (1980) found that four-way broiler crosses exhibit negative heterosis (-5 to -27 percent) while two- and three-way crosses generally had positive heterosis of 1-9 percent. This is an unusual pattern for body weight in chickens that does not correspond to dominance or most epistatic models. The heterosis estimates of Verma and Chaudhry (1980) varied over time (i.e. three-way crosses: 1 percent at two weeks; -12 percent at four weeks; 6 percent at six weeks, etc.) and had large sampling errors.

Heterosis for mature body weight ranges from -3 to 6 percent, but is normally in the range of 2 to 5 percent. As with sexual maturity, heterosis for body weight deviates from expectations resulting from dominance in F_2s. Although dominance is the major genetic mechanism for body weight heterosis, additive x additive epistasis influences heterosis for mature body weight in Leghorn crosses (Fairfull et al., 1985, 1987). In most cases, heterosis for body weight is not desirable in layers as it commonly reduces feed conversion by increasing the maintenance requirement.

Viability. Heterosis for viability is highly complex and variable (-9 to 24 percent), and crosses do not always exhibit heterosis for viability (see Table 37.1).

Heterosis for viability results from a complex interaction of the specific effects of several diseases and stresses. The high variability is likely caused by the effects of different pathogens or strains of pathogens. Thus, the viability or morbidity resulting from each pathogen should be viewed as a separate trait. Unless test conditions are defined in terms of the pathogens present and their virulence, overall viability is not a good trait to elucidate heterosis or other genetic mechanisms and generalizations should be avoided. Under specific pathogen-free conditions, little heterosis occurs so it is likely that heterosis for most traits is affected by pathogens (Gavora, 1988, personal communication).

Other traits. Egg yield, or egg mass which is the total weight of all eggs laid, is the product of egg numbers (production) and egg weight. Heterosis for egg yield is considerable

(5 to 31 percent, see Table 37.1) as expected in a trait heavily influenced by egg production. As for egg production, heterosis for egg yield results either from epistasis or from both epistasis and dominance (Sheridan and Randall, 1977; Fairfull et al., 1985, 1987), so that recombination loss can be expected for this trait. Feed conversion (grams of feed per gram of egg laid) shows considerable heterosis (-16 to 0 percent), but less than egg production traits and egg yield. Also, Australorp x Leghorn crosses generally have higher heterosis for feed conversion (normally -13 to -16 percent) than other crosses (-3 to -9 percent), so that epistasis may be involved in feed conversion heterosis of Australorp x Leghorn crosses. In reciprocal Rhode Island Red x Light Sussex crosses, Damme et al. (1986) found little or no heterosis for fasting metabolic rate, so that very little heterosis for feed conversion likely derives from it.

Feed conversion in broilers (grams of feed per gram of weight gain) exhibits considerable heterosis (7 to 16 percent) when calculated on a constant age basis (Table 37.1). Estimates on a constant weight basis are not available, but it is possible that heterosis would be increased as estimates on an age constant basis penalize rapidly growing birds (Chambers and Lin, 1988).

Fertility is a trait of the parents rather than of the embryo. A hybrid embryo exhibits no heterosis (Table 37.2). A hybrid dam exhibits heterosis for fertility, but a hybrid sire seems to have little effect (Horn, 1974; Fairfull et al., 1987). Unlike fertility, there is heterosis for hatchability made possible by a hybrid embryo (Merritt and Gowe, 1960; Morris and Binet, 1966; Fairfull et al., 1987) and the dam's genotype expresses heterosis. However, as with fertility, the sire's genotype has little effect on heterosis for hatchability.

Miscellaneous. Averages and summaries do not provide a complete picture. Much variation occurs among individual crosses and each genetic base has unique characteristics. The results obtained with the cross of any two strains, no matter how derived, can only be generalized to a limited extent. Although some of the results in Table 37.1 are from the cross of one pair of strains, most are averages representing the crosses of several strains and, as such, much of the variation is hidden. That variation is as valid and of as much interest as variation among experiments. Table 37.3 shows the range of heterosis values in one environment of 24 strain crosses from different genetic backgrounds. Heterosis ranged from -6.1 to 9.1 percent for viability, -1.4 to 15.8 percent for egg production, and -0.4 to 5.9 percent for egg weight. The ranges for viability and egg weight are greater than those reported in Table 37.1. Heterosis for egg production between the crosses of any two strains is not always automatic or always 10 percent or even 5 percent. There is a great deal of variation in heterosis between crosses of pairs of strains and this heterosis depends on the

Table 37.2. Mean heterosis as a percentage of the midparent value of White Leghorns for fertility (FERT) and hatchability (HATC). (Modified from Fairfull and Gowe, 1986; Fairfull et al., 1987).

Genotype				
Sire	Dam	Embryo	FERT	HATC
Pure	Pure	2-strain (F_1)	-1.3	8.9
Pure	2-strain	3-strain	4.1	11.5
2-strain	2-strain	4-strain	3.0	11.9
2-strain	2-strain	F_2	5.6	9.7

nature and degree of the genetic differences between strains.

As can be seen in Table 37.1, the mean performance of two-strain crosses is superior to that of the mean performance of three-strain crosses, which are in turn better than four-strain and F_2 crosses. In layers, economic performance is reduced by recombination loss in the egg production traits. However, Table 37.1 provides an incomplete picture. Means for the seven 'best' of 76 genotypes are presented in Table 37.4. 'Best' is somewhat objective as the rank of the crosses based on income (egg returns plus spent hen value minus feed cost during lay) changes with time and place (market). As can be seen, four of these seven superior genotypes are three-strain crosses, and by any objective measure three of the four best genotypes are three-strain crosses. The individual pure strains and four-strain or F_2 crosses had relatively poor performance compared to the better two-strain and three-strain crosses. So despite a general superiority of two-strain crosses, specific three-strain crosses have excellent performance and the use of three-strain crosses makes commercial sense. Where the performance warranted, the use of a four-strain cross in layers would make sense commercially; however, present evidence indicates that a superior four-strain cross would be hard to find. Note that this discussion does not apply to broilers.

Turkeys

Heterosis for egg production in turkeys is substantial (Table 37.5). These estimates are as large as the highest estimate in chickens (Australorp x Leghorn crosses) and are double the estimates of heterosis for egg production in broilers. Estimates of heterosis for early body weight in turkeys are similar to those in broilers (see Tables 37.1 and 37.5). However, Jerome et al. (1960) estimated heterosis for juvenile body weight to be substantially higher than any other estimate of body weight in turkeys or broilers. Although there are several plausible explanations for the very high heterosis estimates for egg production and juvenile body weight, it may be that epistasis is involved in heterosis for these traits in some turkey crosses. Also, Hassan et al. (1984) reported a mean of 14 percent heterosis for 12- and 16-week body weight in backcrosses as compared to -3 percent in two-way crosses.

In turkeys, there is considerable heterosis for fertility (6-22 percent). The sire's genotype influences heterosis, but the dam's genotype has no effect on heterosis for fertility (Friars et al., 1963). As in chickens, turkey embryos exhibit heterosis for hatchability (2-25 percent). The dam's genotype expresses heterosis, but the sire's genotype has little effect on heterosis for hatchability.

Table 37.3 Range of heterosis values among 24 Leghorn strain crosses as absolute values (mean) and as a percentage of the mean of parental pure strains (midparent value). (Fairfull and Gowe, unpublished).

	High		Average		Low	
Trait	Mean	%	Mean	%	Mean	%
Viability, %	8.1	9.1	1.1	1.2	-5.3	-6.1
Hen-housed egg production	39.9	15.8	16.1	6.4	-3.5	-1.4
Egg weight, g	3.3	5.9	1.1	2.0	-0.3	-0.4

Table 37.4. Means of seven Leghorn strain crosses having superior performance, from a comparison among 4 strains, 12 2-strain, 24 3-strain, 24 4-strain and 12 F_2 strain crosses. (Fairfull, Gowe, and Nagai, unpublished).

Trait	Strain cross						
	1x6	4x6	6x1	1x(4x6)	1x(6x8)	1x(8x4)	1x(8x6)
Viability 134-497 d, %	93.2	89.1	92.8	87.5	91.7	93.7	92.7
Hen-housed production 134-497 d	270	270	273	276	271	270	281
Age at 50% production, d	147	142	148	142	144	140	142
Hen-day rate 134-273 d, %	81.5	86.5	81.2	87.6	80.7	86.1	84.8
Hen-day rate 274-385 d, %	82.6	80.5	81.2	85.0	81.6	78.9	83.5
Hen-day rate 386-497 d, %	67.7	63.9	67.5	67.6	67.5	63.3	68.7
Egg yield 134-497 d, kg	15.8	15.8	16.5	15.9	15.9	15.4	16.3
Egg weight 240 d, g	57.8	57.6	59.4	56.7	57.8	56.2	57.1
Specific gravity 240 d, $(-1)x10^3$	86.4	84.6	85.9	87.1	87.9	87.8	86.8
Haugh units 240 d	86.8	87.4	86.5	87.1	85.4	88.2	85.9
Blood spots 240 d, %[1]	98.7	97.5	97.3	97.1	98.2	99.4	97.7
Egg weight 450 d, g	63.5	63.8	66.1	62.6	63.1	63.4	63.5
Income index, $[2]	9.93	9.60	10.98	10.38	10.43	9.32	10.86

[1] Percentage of eggs free of blood spots.

[2] Income minus feed costs in the laying period only; does not account for pullet cost, fixed costs, labor costs, and so on; costs and prices for Ontario, March 1988.

Table 37.5. Mean heterosis and reciprocal difference as a percentage of the midparent value of turkey stocks for hen-housed egg production (HHP) and body weight (BWT), from the literature.

Reference	HHP	BWT
HETEROSIS		
Two-way crosses		
Friars et al. (1963)	44	$3^E/2^I$
Hassan et al. (1984)		$-5/0^I$
Jerome et al. (1960)		$-2/16^I$
Nestor (1971)		$2^E/0^I$
Backcrosses		
Hassan et al. (1984)		$-15/39^I$
RECIPROCAL DIFFERENCE		
Friars et al. (1963)	47	$3^E/3^I$
Hassan et al. (1984)		$0/10^I$
Nestor (1971)		$10^E/16^I$

[E] Early body weight in weeks 1 to 10.

[I] Intermediate body weight in weeks 11 to 20.

Ducks And Geese

Ducks are maintained for egg production and for meat production. For traits related to egg production, ducks manifest considerable heterosis (Table 37.6). Heterosis for egg production, egg mass, and feed conversion ranges from 7 to 24 percent (Hetzel, 1986). For egg weight and age at first egg, the scale of heterosis is lower at 2 to 4 percent. There is a remarkable parallel with heterosis for similar traits in chickens.

Ducks exhibit considerable heterosis for body and carcass weight traits, carcass composition traits, and tenderness (Table 37.7). In Alabio x Khaki Campbell and Alabio x Pekin crosses, heterosis for many characters is undesirable. It is negative for body and carcass weight traits, and percentage protein, and positive for carcass fat and feed conversion (Hetzel, 1982, 1983, 1986). The magnitude of the negative heterosis for body and carcass weight increased with age from 6 to 16 weeks of age. While other heterosis estimates in meat birds are similar in magnitude, the negative heterosis for body weight and growth in these particular crosses is the opposite of the usual pattern in meat birds. In Pekin x muscovy and Pekin x Small White broiler duck crosses, heterosis for body and carcass characters (positive) and fat percentage (negative) was desirable (Koci et al., 1982; Leclercq and deCarville, 1986). Positive heterosis for subcutaneous fat is desirable in some barbecue markets and was estimated at 3 to 10 percent.

In geese, heterosis for most traits is in an undesirable direction (Table 37.8). In White Italian x Bilgorai crosses, heterosis for dressing percentage and feather yield was negative, and heterosis resulted in poorer feed conversion (Faruga and Majewska, 1982, 1983). In Large Grey x Rhenish crosses and backcrosses, heterosis depressed fertility and hatchability in both the first and second cycles of egg production (Ryabokon, 1984). Similarly, in White Italian x Pomeranian (Karasinski, 1977) and Large Grey x Rhenish crosses, heterosis resulted in lower body weight and reduced feed conversion. In White Italian x Kuban crosses, heterosis for lean weight was positive (Wawro et al., 1985). In Large Grey x Rhenish backcrosses, heterosis for egg production increased substantially and heterosis for body weight and feed conversion was in a desirable direction. These recombination gains indicate that epistasis may be responsible for much of the heterosis in many goose crosses.

In several instances involving weight traits of ducks and geese, two-way crosses had poorer performance than one and often both parental pure strains and backcrosses.

Table 37.6. Mean heterosis and reciprocal difference as a percentage of the midparent value of Alabio x Khaki Campbell (AxK) and Alabio x Tegal (AxT) duck crosses for egg production traits. (Modified from Hetzel, 1986).

	Heterosis		Reciprocal difference
	AxT	AxK	AxK
Rate of lay	7	19	
Survivor egg production	7	24	10
Age at first egg		-4	3
Average egg weight	2	4	2
Egg mass	10	24	10
Feed conversion	-10	-20	12

Table 37.7. Mean percent heterosis and reciprocal difference[1] of ducks for body weight (BWT), carcass weight (CWT), slaughter yield (SYLD), breast meat weight (BMWT), thigh weight (TWT), carcass fat weight (CFWT), feed conversion (CONV), dry matter (%DM), protein % (%PROT), fat % (%FAT), ash % (%ASH), tenderness (TEND) and cooking loss (%CL), from the literature.

Reference	Age	BWT	CWT	SYLD	BMWT	TWT	CFWT	CONV	%DM	%PROT	%FAT	%ASH	TEND	%CL
HETEROSIS														
Alabio x Khaki Campbell crosses														
Hetzel (1986)														
	10[2]wk	-4		0		-17[3]								
	16 wk	-4		-2	-19[5]	1[3]	12				11			
Alabio x Pekin crosses														
Hetzel (1982)														
	6 wk	-3												
	10 wk	-6			-8		25							
	16 wk	-9			-15		0							
Hetzel (1983)														
	6[6]wk	-4	0	1	5	-7		3	2	-1	6	1		
	10[7]wk	-6	-5	0	0	-17		5	12	-8	26	-2		
	16 wk	-8	-13	-2	-2	1		8	-1	-4	0	-1		
Pekin x muscovy crosses														
Leclercq and deCarville (1986)														
	6[2]wk	11		-2	-2[8]	-1[3]					-26[9]			
	10 wk	9		0	14[8]	-4[3]					-48[9]			
	12 wk	10		1	16[8]	-1[3]					-56[9]			
Pekin x Small White crosses														
Koci et al. (1982)														
	7 wk	8	2	1	9	-10	9		-1/1	1/2	-27/-1	13/18	36/56	1
RECIPROCAL DIFFERENCE														
Pekin x Small White crosses														
Koci et al. (1982)														
	7 wk	3	3	0	2	4	17		0/4	3/6	44/51	10/21	3/13	8

[1] Percentage of midparent value. [2] Eight weeks of age for body weight. [3] Thigh plus leg as a proportion of the carcass. [4] Sixteen weeks of age for body weight. [5] Breast muscle proportion at 1.0 kg carcass weight. [6] Eight weeks of age for body weight and feed conversion. [7] Twelve weeks of age for body weight and feed conversion. [8] Breast muscle percentage of the carcass. [9] Abdominal fat.

Table 37.8. Mean heterosis and reciprocal difference as a percentage of the midparent value of geese for fertility (FERT), hatchability (HATC), viability (VIA), egg production (EGGP), egg weight (EWT), body weight (BWT), lean weight (LWT), feed conversion (CONV), dressing percentage (DRES), and feather yield (FYLD), from the literature.

Reference		FERT	HATC	VIA	EGGP	EWT	BWT	LWT	CONV	DRES	FYLD
HETEROSIS											
Two-way crosses											
Italian x Bilgorai crosses											
Faruga and Majewska (1982, 1983)					4	0	-1		3	-1	-8
Italian x Pomeranian crosses											
Karasinski (1977)							-5		9		
Italian x Kuban crosses											
Wawro et al. (1985)							1	4			
Large Grey x Rhenish crosses											
Ryabokon (1984)	- year 1	-3	-3	3	8		-5		6		
	- year 2	-4	-5		-3						
Backcrosses											
Ryabokon (1984)	- year 1	-1	-9	4	21		5		-3		
	- year 2	-1	-4		6						
RECIPROCAL DIFFERENCE											
Italian x Bilgorai crosses											
Faruga and Majewska (1982, 1983)					5	2	2		3	1	0
Italian x Pomeranian crosses											
Karasinski (1977)							2		3		
Large Grey x Rhenish crosses											
Ryabokon (1984)	- year 1	2	11	1	0		6		8		
	- year 2	5	7		3						

Other Poultry

Estimates of heterosis in Japanese quail are consistent with the majority of estimates in chickens (Table 37.9). Heterosis is 11 to 13 percent for hatchability, 2 to 7 percent for body weight, and -9 to 3 percent for sexual maturity (age at 50 percent egg production).

The guinea fowl has superior meat quality and sensory properties. Swedish workers studied chicken x guinea fowl hybrids to estimate the feasibility of combining the guinea fowl's meat quality and the chicken's growth rate (Kiessling, 1978). Heterosis for enzyme activity in the pectoralis major and sartorius muscles was negative (-2 to -52 percent) with two exceptions (both 24 percent). Heterosis for muscle fibre character (diameter, frequency and area) varied widely by muscle and fibre type, but was generally not in a desirable direction. Thus, improvements in meat quality in chicken x guinea fowl hybrids over that of chickens were judged to be minimal.

Chicken x Japanese quail hybrids are completely sterile (-79 percent heterosis for seminiferous tubule diameter) and exhibit negative heterosis (-42 to -9 percent) for skeletal dimensions (Takashima and Mizuma, 1981a, 1981b) resulting in a hybrid closer in size to the Japanese quail than the chicken.

RECIPROCAL EFFECTS

Reciprocal effects are the result of *sex-linked* (genes on the Z chromosome) and *maternal* (confounded by effects of genes on the W chromosome in females and by effects of mitochondrial genes) effects. Sex-linkage probably accounts for most important reciprocal effects, as Z chromosome genes are known to affect egg production and other traits in chickens. Except for early growth rate (Merritt and Gowe, 1965; Al-Murrani, 1978; Aggarwal et al., 1979) and effects of disease mechanisms, maternal effects are not generally considered important (Wearden et al., 1965; Bernon and Chambers, 1985).

In poultry, *paternal effects* are the result of Z chromosome linked genes. Although a true paternal effect caused by semen-transmitted disease or other factors is possible, little evidence supports such paternal effects in poultry and the probability of a true paternal effect is negligible. Maternal effects are confounded with effects of W chromosome linked genes in females. Where both sexes can be measured, maternal and sex-linked effects can usually be separated unambiguously. Some evidence suggests that genes on the W chromosome are responsible for maternal effects for body and egg weight (Lowe and Garwood, 1981; Willeke, 1982). In chicken x Japanese quail hybrids, mitochondrial DNA was maternally inherited (Watanabe et al., 1985), so that mitochondrial genes are a potential source of maternal effects.

Table 37.9. Mean heterosis and reciprocal difference as a percentage of the midparent value of Japanese quail for hatchability (HATC), body weight (BWT), and age at 50 percent egg production (A50P), from the literature. (Adapted from Chahil et al., 1975).

Reference	HATC	BWT	A50P
Heterosis	11/13	2/7	-9/3
Reciprocal difference	1/57	1/11	2/14

Maternal effects are more important to disease and parasite resistance than sex-linkage. Maternal effects have been observed for parasitic challenge with *Eimeria* spp. (Jeffers et al., 1970) as well as for Marek's disease (von Krosigk et al., 1972). The presence or absence of maternal antibody can contribute to a maternal effect for viability (Nordskog and Pevzner, 1977). Also, congenitally transmitted diseases may contribute to maternal effects as has been shown for the lymphoid leukosis virus (Payne et al., 1982; Fadly and Okazaki, 1982; Fairfull et al., 1984).

Reciprocal differences are substantial for viability (1 to 15 percent) and hen-housed egg production (0 to 19 percent) in chickens and for egg production in turkeys (47 percent) and ducks (10 percent) (Tables 37.5, 37.6 and 37.10). One component of hen-housed egg production is viability. Thus, the large reciprocal effect for egg production often may result partially from differences in viability, especially as the reciprocal effect for sexual maturity (another component of hen-housed egg production) is normally much lower. Reciprocal effects for egg yield and feed conversion are substantial in chickens and ducks, but generally lower than corresponding heterosis. Reciprocal effects for sexual maturity and egg weight in chickens and ducks, and mature body weight in chickens, are of about the same magnitude as heterotic effects. In chickens, reciprocal effects for egg quality (egg specific gravity 1-3 percent, Haugh units 1-2 percent, and percentage of eggs free of blood spots 2-5 percent) are larger and more consistent than heterosis.

Reciprocal differences for early body weight in broiler chickens (2-21 percent), turkeys (1-10 percent) and Japanese quail (1-11 percent, Table 37.8) are larger than heterotic effects. In Pekin x Small White broiler duck crosses, substantial reciprocal differences were reported for carcass fat, carcass fat percentage, ash percentage, tenderness and cooking losses (Table

Table 37.10. Mean reciprocal difference as a percentage of the midparent value of poultry stocks for viability (VIAB), hen-housed egg production (HHP), hen-day rate of egg production (HDR), egg yield (EYLD), feed conversion (CONV), age at first egg (AFE), egg weight (EWT), egg specific gravity (SGR), Haugh units (HAU), percentage of eggs free of blood spots (BSP), and body weight (BWT), from the literature[1].

	Two-way crosses[2]						
	A	B	C	D	E	F	G
VIAB	5(1/8)		9(2/15)	14	2	1(0/1)	7
HHP	6(1/19)	1	7(3/10)	4(1/6)	4(0/17)		8(7/8)
HDR	4(3/5)						6
EYLD	3(2/4)		3(2/4)		2(1/2)		4
CONV	1		6(4/8)	3	3(1/5)	2(1/4)	8
AFE	4(1/9)		1(0/2)	3	7		-
EWT	1(0/2)	1	5	2	4(0/12)	1	3
SGR	1	1		1			3(2/3)
HAU	2(1/2)						2
BSP	2						4(3/5)
BWT	3(1/4)^M			0^M	2(0/5)^M	7(2/21)^E	3^M

[1] Damme et al. (1986), Fairfull et al. (1987), Jain and Chaudhry (1985), Orozco and Campo (1975), Sah et al. (1985), Fairfull and Gowe (1986).

[E] Early body weight in weeks 1-10, [M] mature body weight.

[2] First egg-production cycle: A=Leghorn crosses, B=Rhode Island Red (RIR) crosses, C=Australorp x Leghorn crosses, D=Leghorn x RIR crosses, E=other egg-type crosses, F=meat-type crosses; second egg production cycle G=Leghorn crosses.

37.6) that (except for tenderness) were equal to or greater than heterosis for these traits.

In geese, reciprocal differences were considerable for fertility, hatchability, egg production, body weight, and feed conversion (Table 37.8). There are considerable reciprocal differences for fertility (0-15 percent) and hatchability (0-6 percent) in turkeys although these are substantially lower than corresponding heterosis (Friars et al., 1963).

In many cases, reciprocal differences are taken for granted. In broiler chickens, the reciprocals of commercial crosses are not considered, given the performance and function of the parental crosses. However, reciprocal differences should not be forgotten.

FACTORS AFFECTING HETEROSIS

Environment. The magnitude of heterosis can be influenced by environment (Barlow, 1981). In poultry, changes in heterosis have been found with respect to location, nutritional treatment (Hull et al., 1963; Emsley et al., 1980), housing (Blyth and Sang, 1960; Horn et al., 1980; Gowe and Fairfull, 1982a), hatch (season, year) and temperature (Chahil et al., 1975; Al-Soudi and Al-Jebouri, 1979).

In two recent studies using high performance stocks in two cycles of egg production, Rhode Island Reds housed two and four hens per cage (Horn et al., 1980) and White Leghorns housed one and three hens per cage (Gowe and Fairfull, 1982a; Table 37.11), heterosis for egg production traits enlarged and heterosis for body weight decreased as the stocking rate increased. For viability, egg production, and feed consumption of White Leghorns, Emsley et al. (1980) found consistently higher heterosis (3-5 percent) on a 13 percent protein than on a 17 percent protein diet. Using White Leghorns, Hull et al. (1963) found heterosis for several traits varied with location in each of four years. There were heterotic differences of up to 3 percent between full-fed and restricted feeding regimens for age at first egg and housing body weight in some years on each farm, but these did not consistently favor one regimen. Al-Soudi and Al-Jebouri (1979) found heterosis for feed consumption was 8 percent higher (14 vs. 6 percent) in a cold than in a hot environment with Leghorn x Iraqi crosses. In Japanese quail, large changes in heterosis occurred with respect to hatch (hatchability) and heat stress (body weight) (Chahil et al., 1975). Also, it is not unusual for heterosis to vary from year to year at any location (i.e. Hull et al., 1963; Singh et al., 1980). Generally, but not invariably, heterosis increased with the severity of the environment.

Environment affects reciprocal differences also. Reciprocal differences for viability (133-496 days of age) and hen-day rate of egg production were greater with three hens per cage (10.2 cm feeder and 464.5 cm^2 floor space per hen) than with one hen per cage (20.3 cm feeder and 929.0 cm^2 floor space per hen) (Gowe and Fairfull, 1982a; Fairfull et al., 1983; Table 37.11). For mature body weight, the trend was reversed; reciprocal effects were larger for hens housed one per cage. Reciprocal differences for viability and hen-housed egg production were larger (3-5 percent) and in the opposite direction with a 17 percent instead of a 13 percent protein diet (Emsley et al., 1980). Environment affected reciprocal differences with respect to hatch for hatchability and heat stress for body weight (Chahil et al., 1975).

Correlations between mean performance of strain-cross hens housed one per cage and three per cage are moderate to high for hen-housed egg production and egg weight, but lower for viability (Fairfull and Gowe, 1986). For hen-housed egg production, Horn (1982) reported genetic correlations between hens housed two and four per cage of 0.69 in the first cycle and 0.72 in the second cycle of egg production. These values reflect changes due to

Table 37.11. Mean percentage heterosis and mean percentage reciprocal effect at two stocking rates[1] in first and second laying cycles. (Fairfull and Gowe, unpublished).

	Heterosis Age in days				Reciprocal effect Age in days			
	133-496		546-909		133-496		546-909	
Trait	1/c	3/c	1/c	3/c	1/c	3/c	1/c	3/c
Laying viability	1	1	1	7	4	8	7	7
Hen-housed egg production	6	8	1	1	4	3	7	
Age at 50% production	-3	-3			2	2		
Hen-day rate of lay	6	7	16	9	3	5	4	8
Body weight	4	2	5	2	4	2	3	3
Egg weight	2	2	2	2	2	2	3	3
Egg specific gravity	0	0	0	0	0	0	0	0
Haugh units	1	0	1	1	2	2	2	2
Blood spots	1	1	2	2	2	2	5	3

[1] Stocking rate: 1/c = one hen per cage, 3/c = three hens per cage.

heterosis and reciprocal differences as well as other genetic and environmental effects. While these correlations indicate that the better crosses tend to have superior performance in both environments, changes in rank of crosses in the two environments occur and probably increase with very high density environments.

Age. Heterosis for some traits is affected by age during early growth (Kanavikar et al., 1978; Aggarwal et al., 1979; Roy and Kumar, 1981; Hetzel, 1982, 1983; Jain et al., 1982), and over production cycles (Horn et al., 1980; Gowe and Fairfull, 1982a). In early broiler growth, heterosis for body weight increases from about 0 percent at hatch to a range of 2-10 percent by slaughter age (6-10 weeks). In broiler ducks, body weight heterosis increased from -3 percent at six weeks of age to -8 to -9 percent at 16 weeks and a similar trend was apparent for carcass weight (Table 37.7).

Heterosis for viability (0-6 percent), hen-housed egg production (5-10 percent), and hen-day rate of lay (2-10 percent) increased substantially in a second cycle of egg production compared to the first. Changes in heterosis for mature body weight and percentage of eggs free of blood spots were small but significant (Gowe and Fairfull, 1982a; Table 37.11). Fairfull et al. (1987) found heterosis for rate of lay increased late in the first laying cycle, but this was the opposite of the trend found in earlier work (Fairfull et al., 1983). Heterosis for egg yield and feed conversion increased progressively through the first laying cycle (Fairfull et al., 1983). However, changes within laying cycles are relatively small compared to changes between laying cycles.

Changes in reciprocal effects over production cycles reflect those of heterosis, but are of lesser magnitude. Reciprocal differences for viability (1-3 percent), hen-housed egg production (3-4 percent), hen-day rate of lay (1-3 percent) and percentage of eggs free of blood spots (1-3 percent) in a second cycle of egg production were lower than in the first cycle (Gowe and Fairfull, 1982a; Table 37.11). In Black Bronze x Ross White turkey crosses, reciprocal differences increased by 6 percent from 12-16 weeks of age (Hassan et al., 1984).

Correlations of strain-cross means are substantial between first and second cycle performance ranging from about 0.5 for viability to about 0.9 for egg weight with three hens per cage (Fairfull and Gowe, 1986). However, imperfect correlations suggest changes in ranking of the crosses and, in fact, substantial changes do occur (Fairfull, 1982). The better crosses commonly have superior performance in both cycles of egg production and the genetic effects important in the first cycle were important also in the second cycle. Despite these facts, although the breeder can probably safely discount crosses with poor first cycle production, the performance of the better layer crosses intended for markets where the birds are molted for extended use must be tested, and economic value must be determined over more than one cycle of production.

Selection. Cross performance can be improved genetically by developing inbred lines and then testing and retaining the better nicking lines, by using reciprocal recurrent selection to maximize heterosis, and by selection within lines and then testing and retaining the better of the improved lines. Each of these methods with variations has been or is being used by international breeding companies. It is likely that most commercial stocks are improved by selection within strains with a few still using a form of reciprocal recurrent selection. However, if selection based on cross performance is used, it is now likely modified to include a strong within-strain selection component.

Bell (1982) reviewed selection for heterosis and presented the results of a commercial program of reciprocal recurrent selection with egg stocks. Reciprocal recurrent selection results in both increased heterosis and improvement in overall performance. Orozco and Campo (1975) speculated that reciprocal recurrent selection may be better than within-strain selection and crossing of the selected lines either when traits with nonadditive effects are important or when additive genetic variation is nearly exhausted. Although it is theoretically possible for genetic variation to be exhausted, Gowe and Fairfull (1985) reported genetic variation for egg production and other traits essentially unchanged through 30 generations of selection, and in broilers, despite extreme selection for growth, genetic variation does not seem to be greatly diminished. Because nonadditive genetic variation is important for egg production traits, layers should be a good model in which to compare reciprocal recurrent selection to within-strain selection and crossing of the selected lines.

Reciprocal recurrent selection has been compared to within-strain selection in poultry (Saadeh et al., 1968; Calhoon and Bohren, 1974), but the results were inconclusive. This finding is not surprising as both programs have been used commercially without a significant advantage, as far as can be determined, to the companies using them, in terms of the performance of the commercial product. However, within-line selection programs are cheaper to run and the pure strains that result usually have better average performance than pure lines selected for cross performance.

The long-term within-line selection (WLS) studies of Hutt and Cole (Cole and Hutt, 1973), and Gowe (Gowe et al., 1973; Gowe and Fairfull, 1985) illustrate within-strain selection and its effect on crossing. In both studies, gains resulting from heterosis were significant in any generation, especially for egg production traits. Genetic improvement was substantial in the pure strains as a result of selection that accumulated over several generations, and after several generations it was greater in magnitude than heterosis for any trait. In both studies, heterosis remained relatively constant. There were no indications of increased or decreased heterosis in two-strain crosses of unrelated strains. This finding has been corroborated by several shorter-term studies of three to five generations (Kolstad, 1980; Liljedahl and Weyde, 1980; Ayyagari et al., 1982) and similarly for reciprocal recurrent selection (Saadeh et al., 1968). In absolute terms, heterosis among crosses of several WLS strains did not decrease as parental performance increased in either the first or second laying cycles (Fairfull et al., 1983). Thus, heterosis is particular to a specific cross. There is no trend for heterosis to be higher or lower on average in crosses of WLS strains with superior pure-line performance, which is true within or across generations and laying cycles. In certain crosses, heterosis can increase significantly over generations of WLS (see Gowe and Fairfull, 1982b), but this phenomenon is not general.

Because of minor differences in WLS that result in substantial heterosis, genetic separation can accumulate rapidly in lines from a common genetic base. Gowe's study included three pairs of WLS strains derived from common base populations. Each pair was selected for the same battery of traits with the exception that one strain of each pair was selected for hen-day rate of lay from first egg and the other strain for hen-housed egg production. When the related lines were crossed after only six or seven generations of selection, heterosis (4-5 percent in first cycle and 6-8 percent in second cycle) and reciprocal differences (3-6 percent in first cycle and 14-15 percent in second cycle) were substantial for egg production.

New approaches to the exploitation of nonadditive genetic variation need to be proposed and tested. Kinghorn's (1986) proposals for selection across breeds, and Sheridan's (1986a, 1986b) heterosis inheritance backcross evaluation technique for situations where at least one of the parental lines no longer responds to selection, are creative proposals for two situations. Many more attempts need to be made.

USE OF HETEROSIS AND OTHER CROSSING EFFECTS IN BREEDING

The commercial cross is usually a three- or four-way cross. Occasionally, two-way crosses are used. Because the number of parents of commercial birds (parent stocks) required is much greater than the number of selected pure-strain birds, the crossing process is used to multiply the grandparent stocks (increase the number of breeding parents of the commercial bird), so that no apparent 'cost' is attributable to the crossing process more than that required for any other practical system. To produce the commercial bird in any case would require one or two generations of relaxed selection — producing parent stocks from grandparent stocks, and producing commercial broilers or layers from parent stocks.

Heterosis more than offsets any expected differences in performance produced by selection gains in one or two generations. There is little evidence of appreciable reductions in performance because of relaxed selection in layers, although slight declines in performance in the order of 1-3 percent per generation occur (Moultrie et al., 1956; Shoffner and Grant, 1960; Bohren and McKean, 1964; Nordskog and Giesbrecht, 1964). However, as discussed in the section on reciprocal effects, this finding could result from disease rather than genetic change *per se*. The incidence of some congenitally transmitted diseases, such as LLV, increases because of relaxed selection (Gavora and Spencer, 1985), and performance is reduced more in hens congenitally infected with LLV than in horizontally infected hens (Payne et al., 1982; Fadly and Okazaki, 1982; Fairfull et al., 1984). Thus, some losses in economic performance would be expected with a substantial rate of infection of some congenital diseases. Most of the studies mentioned were carried out at a time when mortality losses were much higher than they are now, and before effective treatments or vaccines were available for many poultry diseases that are now under much better control (eg. Marek's disease).

The crossing process is used for two main reasons: firstly, to take advantage of heterosis, and secondly, to combine stocks in which the battery of performance traits complement each other (or conversely to cover particularly poor performance of a pure strain for a commercially vital character, such as poor shell quality). The cross used is not the cross with the greatest heterosis, but the cross with the best commercial performance (which is subtly different from the cross with the best economic performance). The improvement of the cross over the parental pure-lines consists of the heterosis (as presented earlier) plus half of the reciprocal difference where reciprocal crosses were used to estimate heterosis.

In commercial poultry breeding, heterosis is useful at several levels: in the commercial bird, and if they are crosses, in the parent stock dams and sires for several traits (Table 37.12). The list in Table 37.12 is of traits for which useful heterosis is exhibited at each level, rather than being a list of important traits. Other traits, such as egg quality in commercial layers, and male reproduction in male parent stocks of layers or broilers, are important. Also, other traits may express considerable heterosis, such as growth or body size in broiler parent crosses, but this heterosis may not be desirable or useful at that level and may, in fact, create considerable problems.

Complementarity has two aspects in poultry, being variations on a similar theme. One aspect is the use or development of specialized sire and dam lines, which is especially applicable to meat-type poultry. Where the functions of dam (reproduction) and progeny (meat production and quality) can be separated, such as is possible in meat production, the efficiency of the system can be maximized by using specialized sire and dam lines (Smith, 1964). This efficiency is achieved in part by maximizing selection pressures and genetic gains produced by additive effects.

Commercial broiler breeders are using an approach of specialized sire and dam lines. The basic genetic backgrounds of broiler sire and dam lines differ; White Cornish are used for sire lines, and White Plymouth Rock for dam lines. These genetic backgrounds are complementary and fit the criteria of specialized sire and dam lines; as well, the battery of traits selected and the selection emphasis differs in sire and dam lines. For more detail, consult Chapter 40 on commercial breeding practices.

Table 37.12. Level and source of useful heterosis in poultry breeding.

Level	Layers	Broilers
Commercial cross	Viability[1] Egg production Egg size Feed conversion Sexual maturity[1] Hatchability	Viability Growth Feed conversion Hatchability
Parent Stock		
Dam cross	Viability Fertility Hatchability Egg production Feed conversion Sexual maturity	Viability Fertility Hatchability Egg production Feed conversion Sexual maturity
Sire cross	Viability	Viability Freedom from crippling

[1] Affects egg production.

The other aspect of complementarity is combining the strengths of lines, which although it is intrinsic in the approach of specialized sire and dam lines, it is also applicable to layers. Many commercial pure lines do not have equally superior performance for all traits of commercial merit (economic merit plus biases of consumers or producers) and many may have a few truly outstanding characteristics, but could not be used commercially as pure lines because of other failings. For many characters, crosses have performance equal to or better than the superior parent, and for the few others, crosses are equal to or better than the average of their parental pure lines. Thus, when the right combination of parental pure strains is chosen, the strengths of the pure strains are usually enhanced in their crosses and weaknesses in one parental stock magically disappear. In this way, the strengths of parental pure lines that might not otherwise be exploitable, not only can be used to advantage commercially through crossing, but they also can be combined.

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Chapter 38. GENETIC CONTROLS IN SELECTION

R.S. Gowe and R.W. Fairfull

As Hill (1972a) so clearly stated, "the separation of observed change into its environmental and genetic components is an important part of the analysis of selection experiments or breeding programmes". Nevertheless, the practitioners of early poultry breeding research and early commercial breeding programs did not see any need for genetic control procedures. The assumption must have been that any progress made had to be due solely to the selection program (see Hutt, 1949, and Lerner, 1950, for a review of these early selection experiments by Gowell, Pearl, Goodale, Hays, Dryden and others). Even though Lerner (1950) belatedly recognized the need and value of genetic controls, he had no adequate control on his well documented egg production selection study.

In Hutt's (1949) *Genetics of the Fowl*, genetic controls or control strains are not mentioned, but the need for control strains is recognized in a later book (Hutt and Rasmusen, 1982). In their own classical study, Hutt (1949) and Cole and Hutt (1973) used the technique of selecting in opposite directions, known as 'two-way' or 'divergent selection', for resistance and susceptibility to the 'leukosis complex' in Leghorn strains. In addition to divergent selection for resistance to the 'leukosis complex', they selected both lines for other economic traits such as egg production and egg size with varying emphasis in the resistant and susceptible strains. Falconer (1981) pointed out how such a divergent or two-way selection technique can improve the accuracy of estimation of the specific response being measured. However, he also noted that if the interest is primarily in the change in one direction (i.e. resistance in this case), the use of an unselected control is preferable since with two-way selection the response is often not equal in the two directions. Therefore the important long-term study of Cole and Hutt (1973) would have been even more valuable if unselected genetic controls had been maintained to separate environmental trends from genetic trends for a study that took place over many years with many environmental changes.

Theoretically, genotype x environment interactions may preclude the separation of genotypic and environmental trends and have been advanced as a major disadvantage to the use of control strains. Yet it should be realized that without the existence of control strains, there is little or no chance that major important genotype x environment interactions would be known or detected. It is unlikely that minor genotype x environment interactions of limited importance would be detected by any system. Yet there are many that can be detected and estimated providing a control strain exists. A major change in the past decades has been the shift from floor pens to cages for rearing and housing egg stocks. It is doubtful if the effect this important change has had on different genotypes would be fully known without the existence of control strains (Gowe and Fairfull, 1985; Dickerson and Mather, 1976). The control was valid before and after the environmental change, and through the use of the control the size of its effect was estimated. Some types of retrospective evaluation would be idle speculation without control strains. There is little empirical evidence that genotype x environment interactions are a serious consideration in the practical use of control strains.

THEORY AND DESIGN OF CONTROLS

The three major functions of genetic control populations are:

1. To assess the magnitude of short-term fluctuations in the environment and to furnish a means of correction.
2. To maintain genetic constancy over a long period of time, thereby enabling the evaluation of long-term trends in the environment and permitting the estimation of genetic trends in selected or experimental populations.
3. To serve as gene pools and stable base populations, usually with known genetic parameters and frequently with many other known biochemical, molecular and physiological parameters (Gowe et al., 1959b).

It should be noted that the repeat mating method of separating genetic and environmental trends (Goodwin et al., 1960), to be discussed later, does not serve the last important function at all. In research programs on selection, and in applied breeding programs, it is important to be able to separate the genetic changes resulting from the selection practiced from both the affects of short-term environmental fluctuations and long-term environmental trends.

The theory and practice for methods applicable to species where it is necessary to analyze field records to measure genetic changes does not have much utility for poultry, and will not be dealt with further here. Those interested should consult Smith (1962).

The general theory and design of genetic controls has been addressed by several authors and the reader is referred to these sources for expanded discussions on the subject (Gowe et al., 1959b; King et al., 1959; Goodwin et al., 1960; Dickerson, 1969; Hill, 1972a, 1972b; Bray et al., 1962; James, 1962; Latter, 1959; and Sellier, 1980).

An elaboration on the theory of control strains for the special circumstances when there are overlapping generations has been discussed by Cassuto et al. (1970), King and Smith (1982), and Smith (1976, 1977). Overlapping generations can be used to estimate or remove environmental trends. A good example is the use of Best Linear Unbiased Prediction (BLUP) techniques (Henderson, 1984) in estimating genetic change in dairy cattle. Even with discrete generations, BLUP can estimate genetic change using additive genetic relationships between parents and progeny, providing there are no large environmental influences to confound the estimate. Overlapping generations are not common in poultry research or in applied breeding with poultry, although the techniques for dealing with overlapping generations may have some application in industrial broiler breeding and in turkey and goose breeding. In the broiler breeding industry, often breeders of two or more ages are used concurrently and the progeny are reared together. However, breeders of different ages may not be mixed, so that ties between generations would be minimal. Breeder age would be completely confounded with other effects, such as selection (older birds may be selected for more traits or reselected on other records, e.g. progeny) or age, which especially with respect to egg size is well known to have large effects on broiler growth. Although similar in concept to a scheme using repeat matings (Goodwin et al., 1960), it is not directly comparable. This subject will not be dealt with further, but the references listed above should be consulted if the particular circumstances of an applied poultry breeding program or experiment lead to the use of overlapping generations.

METHODS FOR REMOVING ENVIRONMENTAL TRENDS

There are several methods to remove environmental trends and fluctuations from poultry selection and breeding experiments so that the genetic trends can be evaluated. All have their weaknesses and strengths, but a few are superior to others.

Constant environment. If it were feasible to maintain a constant environment (feed, temperature, humidity, light, etc.), genetic change could be measured directly. However, this is only possible under very special laboratory conditions. Generally it will be very expensive if there are large populations, and if all environmental factors are totally controlled. This approach is practical only with laboratory pilot animals such as *Drosophila*, *Tribolium*, mice, and possibly Japanese quail. Even with these small species it is difficult, if not impossible, to maintain constancy in all the environmental elements. This procedure is not recommended for large poultry species because of this inherent problem. In chicken experiments, even when attempts were made to control many elements of the environment, there remained very large environmental effects (Gowe et al., 1959a).

Inbred lines. These can be maintained and used either directly or as crosses of two or more lines. If inbred lines are highly inbred they should be more or less genetically stable. The problem is that highly inbred stable lines are genetically unique and even crosses of such lines represent only a small unique sample of the possible genes of the species or strain. Such populations have a much higher probability of interacting with the environment in a way that is unique to that particular inbred genotype. That is, genotype x environment interactions are much more likely. Inbred lines tend to be more sensitive to environmental fluctuations, so that there are differences between the inbreds and the experimental material in scale of response; there may be an environmental response in the inbred lines without a corresponding response in the experimental lines. Also, if the inbred lines are not very highly inbred, they can drift genetically very quickly without special precautions for prevention; even with precautions they can drift more readily than noninbred populations. See Hill (1972a) and King et al. (1959) for a more detailed discussion of why inbreds and inbred crosses usually do not make good control populations.

Divergent selected strains. If the only objective is to measure the regression of response on the selection differential, then two divergently selected lines will result in the most efficient use of resources (Falconer, 1981). A recent report by Leclercq (1988) illustrates how this procedure can be used successfully when the principal objective of the research is to establish two contrasting lines for further physiological and biochemical studies. However, as has been pointed out already, this is not the most useful method if the experimentor has a major interest in selection response in one direction, and it also is certainly not a good method if the absolute genetic trend is of importance. The latter is the usual situation in commercial breeding programs.

Comparison of selected strains. The comparison of responses in different lines being selected in the same direction, but with different selection procedures or from different genetic base populations, is an alternative used by some researchers. This avoids using space for a separate control population. While it does permit a comparison among the breeding systems being tested, it does not permit an evaluation of the absolute magnitude of the different responses. The limitation here in practical breeding programs is that the absolute change is usually critical in evaluating the usefulness of the different selection procedures being compared. It is essential to determine if the selection procedures being tested are actually resulting in positive genetic progress in the traits under selection. Some studies have revealed negative genetic progress in some selected populations (e.g. Calhoon and Bohren, 1974; Nordskog et al., 1974), and without control strains this would not have been known.

Unpedigreed random breeding control. Perhaps the simplest type of control is just to maintain a random breeding population without selection that has been carefully sampled from the base population. If the population is large, and reasonable care is taken in sampling it from generation to generation, genetic changes will be small. It has the advantage of being relatively economical to maintain for naturally mated species, particularly if the progeny produced can be used in some practical way between reproductions. Nevertheless, under most circumstances it is more efficient and also more economical to maintain a pedigreed randombred control to be described next. Also, the pedigreed controls provide more information on genetic parameters which are usually of interest.

Before leaving the unpedigreed randombred population, some further comments are in order. Random breeding populations that are not pedigreed can be artificially inseminated, so that each male is mated to the same number of females when they are reproduced. This will reduce the variation in progeny per parent below that which would be expected if there was natural flock mating. There is also a greater degree of assurance that most of the parents contribute progeny to the next generation. As a result, genetic drift will be reduced when compared to a truly random breeding population (Crow, 1954). In general, the effective number of parents in the random nonpedigreed situation is given by:

$$1/N_e = 1/4M + 1/4F$$

where N_e is the effective population size and M and F are the number of males and females mated in the random breeding populations. The N_e for a random breeding population will change (increase) if there are restrictions such as artificial insemination, as discussed above. However, the changes in N_e will likely be small. Appropriate formulae to estimate N_e under mating plans where the variance in family sizes was reduced were developed by Latter (1959) and were discussed by Gowe et al. (1959b) and Hill (1972a).

Pedigreed random breeding control. A pedigreed randombred control strain is one that is reproduced each generation with full pedigree information. To minimize genetic drift with a pedigreed control, the mating plan is structured so that each male provides a male, and each female a female, to reproduce the next generation. By the use of artificial insemination it is possible to use a ratio of one male mated to one or more females, depending on the use and other needs of the control strain. The closer the ratio of males and females is to 1:1, the more efficient is the system in reducing inbreeding and genetic drift for a given total size of control population. Sampling among individual parents is eliminated in the pedigreed control strain if there are equal numbers of males and females, and minimized compared to random unpedigreed populations if there are more females than males. In the latter case, there is only sampling among females to produce the males (only one of the females can provide the male progeny used as a breeder) to breed the next generation. With this latter mating plan the effective number of parents (Gowe et al., 1959b) is given by:

$$1/N_e = 3/16M + 1/16F$$

In Table 38.1 the effective numbers of parents and the change in the inbreeding coefficient (F) per generation with different numbers of male and female parents are compared.

For both the pedigreed and unpedigreed random breeding controls it is critical that these populations are randombred for at least two, and preferably three, generations to bring the population into equilibrium before their use as a control strain. This is particularly important if highly selected strains or composites (synthetics) of highly selected strains are used to develop a control. This will allow the epistatic combinations produced by the selection programs to break up and for the genes to come into equilibrium.

Frozen embryos. Embryos can now be stored indefinitely in liquid nitrogen for some species. It is not practical at the present time with bird embryos, and it is unlikely to be practical in the near future because of the very large ovum and the advanced development of the embryo when the egg is laid. The technique of deep-freezing embryos from the base or control population, to be compared with selected populations many generations later, is therefore not now available to poultry scientists. If the procedure does become available, its value for maintaining control strains will depend to a great extent on the procedure resulting in a minimum loss of embryos, so there is little or no selection for freezability which could make the stored embryos less representative of the original gene pool.

Frozen semen. It is now possible to store frozen chicken semen for long periods. This makes it practical to store semen from a random sample of the base population males. At the end of the selection study, or periodically throughout, this semen can be used to mate a sample of the selected population. The resulting progeny would be expected to have half the genetic progress of the selected lines, and thus permit estimates of the genetic gain. One major disadvantage of this procedure is that semen from all males does not freeze equally well and the semen of a significant proportion of males seems impossible to freeze with present techniques (Ansah, 1988, personal communication). It is also quite

Table 38.1. Effective number of parents for two mating plans, random breeding (R) and pedigree random breeding (P), and the inbreeding expected with flocks of different sizes. (From Gowe et.al., 1959b).

Actual parents		Mating	Effective number	Change in inbreeding coefficient
Males	Females	plan	of parents (N_e)	(F) per generation, percent
25	25	R	50	1.0
50	50	R	100	0.5
100	100	R	200	0.2
20	200	R	73	0.6
50	250	R	167	0.3
80	240	R	240	0.2
100	300	R	300	0.1
25	25	P	100	0.5
50	50	P	200	0.2
100	100	P	400	0.1
20	200	P	103	0.4
50	250	P	250	0.2
80	240	P	384	0.1
100	300	P	480	0.1

possible that there are genetic correlations between the freezability of the semen of different males and other important traits. If there are, this type of control will be biased. As techniques for freezing improve and a higher proportion of male's semen can be successfully frozen, this bias should be reduced. In addition, there is the distinct possibility of heterosis between the selected and unselected or original genotypes. Fairfull et al. (1983b) found that after dividing populations, followed by only six or seven generations of similar selection in both lines, there was substantial heterosis for egg production between the crosses of strains from the same origin. Heterosis is not usually considered important with this type of control. However, these results suggest that it is a distinct possibility, and it would bias the evaluation of the selection program.

Repeat mating control. In its simplest form, this technique requires mating all or part of the selected males and females again so that their progeny can be compared with the progeny of the next generation of selected birds. However, age effects of the parents would be confounded with the two progeny groups in this simple plan. To correct for this, Goodwin et al. (1960) proposed a repeat mating design that allowed comparisons of progeny of selected parents of the same age, but separated by one generation of selection. This approach has the major advantage of comparing selected and controls that are only separated by a generation; therefore, there would be little likelihood of any genotype x environment interaction. It also permits a practical breeder to keep his controls highly selected so that these birds could be used commercially.

The major disadvantages are the added complications of maintaining populations of different ages and the potential for disease problems. Also, this type of control is not efficient when used with unrelated selected strains, necessitating maintaining a control for each selected population.

Another serious deficiency is that the genetic sampling errors of each generation are cumulative, which is not the case with either of the random breeding control populations (Giesbrecht and Kempthorne, 1965). Flock (1988) pointed out the difficulty in practice of using even a simplified repeat mating control, and stated that he had discarded the use of this control in his commercial program.

As pointed out earlier, the repeat mating control does not preserve the original gene pool so that the breeder can return to it if needed for genetic variation to meet new situations. Also, the scientist does not have a representative original population for use in measuring traits not considered important, or which were impossible to measure, when the selection study was started.

Summary. The most widely useful methods of maintaining genetic controls are the naturally mated (or artificially inseminated unpedigreed) random breeding population or the pedigreed random breeding population. The latter under most conditions is the most effective, efficient, and economical type of control strain to maintain. However, it is important that there be about 250-400 effective parents for stability and minimum drift when used as a control in long-term selection studies.

SOURCES OF ERROR IN ESTIMATING GENETIC CHANGE

The main sources of error in estimation of genetic change from generation to generation using control strains are drift variance, measurement errors, genotype x environment interaction, and relaxed selection effects from nonadditive genetic variation.

Drift variance. This accumulates over generations and arises because of the finite size of the control population. It can be predicted from the N_e of the control population. The change in the mean of a population due to drift is given by the expression V_A/N_e for each generation, or $2FV_A$ for several generations, where F is the inbreeding coefficient and V_A is the additive genetic variance. Since the pedigreed random breeding control has a higher N_e than the unpedigreed random breeding control for the same population size, drift variance is always lower for the former.

It should be noted that for a repeat mating scheme where the repeat-mated control is the selected line itself one generation back, there is no accumulation of drift. However, drift accumulates when such a control is used as a control for an unrelated line. Also, the structure of a repeat mating control using selected parents usually will have a lower N_e than for a pedigreed randombred control, and therefore, drift variance will usually be higher (for methods of computing drift see Hill, 1972a, and Dickerson, 1969).

Measurement errors. These arise from the limited number of individuals recorded each generation. The more individuals measured each generation the lower will be the error in estimating the mean. These errors do not accumulate over generations except for the repeat mating scheme (Hill, 1972a; Giesbrecht and Kempthorne, 1965). This is a serious drawback of the repeat mating control scheme.

Genotype x environment interaction. There is much evidence to show that genotype x environment interactions are not generally of much practical concern where reasonable precautions are taken to ensure that there is not an extreme environmental situation (Hull and Gowe, 1962). However, it is well known that strains can be genetically very different in their susceptibility to specific diseases, and interactions will occur under some disease exposure conditions. Nevertheless, the same extreme disease exposure is not likely to be experienced generation after generation. The use of the regression estimator of genetic change over generations will minimize this error.

The repeat mating control is less likely to be affected differently by different environments than the selected lines since the control and selected lines are only separated genetically by one generation.

Nonadditive genetic variance. Relaxed selection effects are not a practical problem with most methods of correcting for environmental variation. The nonadditive genetic variance of control strains does not bias genetic gain estimates, unless the control strain is a cross or synthetic that was recently formed (within three generations), or a highly selected strain that has not been randomly bred for two or three generations. Relaxed selection effects are of limited duration and most are dissipated after the first generation of relaxation (for further discussion and an example see Chambers et al., 1984). Relaxed selection effects do not apply to the following control procedures: constant environment, inbred lines, divergent selection, frozen embryos, or frozen semen, nor to randombred control strains after three generations of relaxation.

HISTORY AND USE OF CONTROL STRAINS

In this section the use of control strains with various domestic bird species will be reviewed. It is not intended to be a complete review of all studies that involved a control

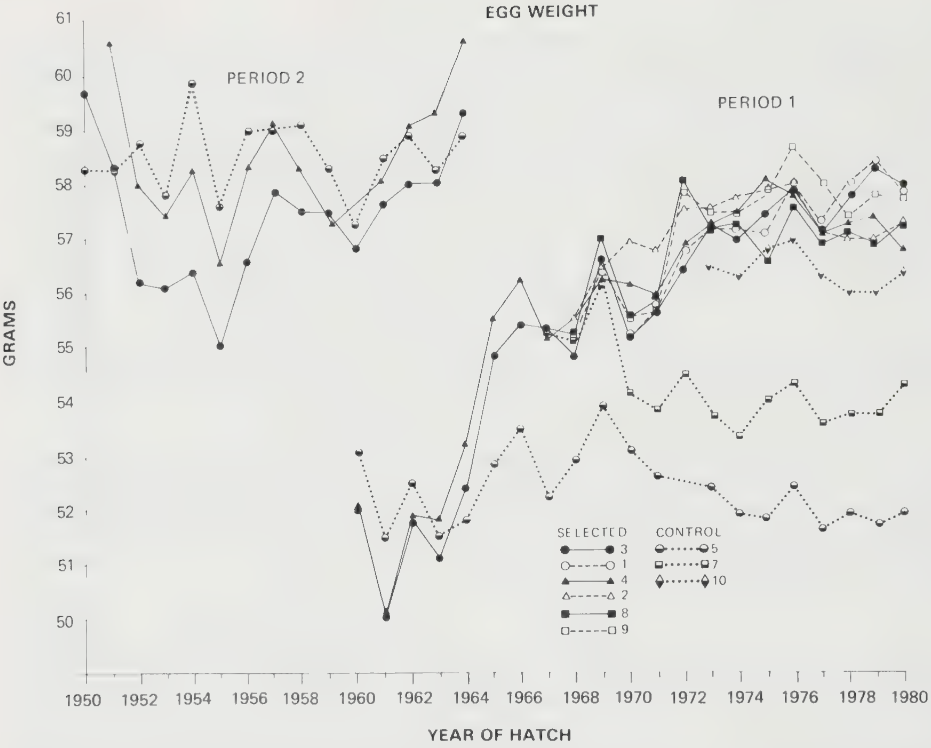

Figure 38.1. Survivor egg weight for selected Strains 1, 3, 2, 4, 8 and 9, and unselected control Strains 5, 7 and 10. Measured for Strains 3, 4 and 5 at about 350 days (Period 2) up to 1964, and for all strains at about 225 days from 1960 to 1969 and about 240 days from 1970 to 1980 (Period 1).

strain. Rather it will deal with some of the reports that have added to knowledge and experience in how to maintain and use controls, and it will provide examples of their use with the major domestic species.

Chicken Egg Stock Control Strains

Australia: Commonwealth Scientific and Industrial Research Organization (CSIRO), Sydney. Probably the first use of a control strain to separate genetic and environmental trends in a poultry selection study was the egg stock selection experiment started by Dr. Fred Skaller at the CSIRO Poultry Research Centre in Australia. This first White Leghorn control was started in 1947 (Skaller, 1956). By 1957 inbreeding had risen to F=16.2 percent. Only eight sires were used per generation as late as 1955, although Morris (1963) increased the sire numbers for this random breeding control to 12 from 1956 to 1958, and he again increased the sire numbers to 24 in 1959. Despite its limitations, this first control strain was very useful in helping these authors interpret their experiments. The more recent egg stock program by the CSIRO group employed a pedigreed control that consisted of 40 males and 160 females, at a mating ratio of 1:4 where each male contributed a male and each female a female breeder to the next generation (Yoo et al., 1983).

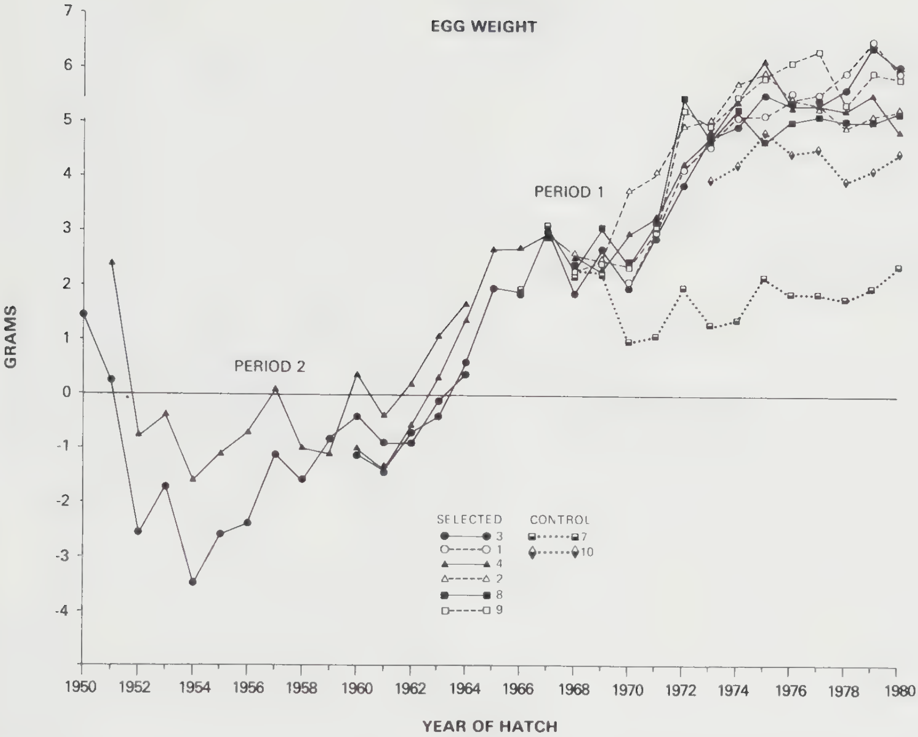

Figure 38.2. Survivor egg weight data from Figure 38.1 expressed as deviations from the unselected control Strain 5.

Canada: Agriculture Canada (AC), Animal Research Centre (ARC), Ottawa. Shortly after Skaller started his control, Gowe in Ottawa, Canada started a randombred White Leghorn control (originally called Ottawa Control Strain, later Strain 5) in 1950 that exists today at that institution after nearly 40 generations of random breeding (Gowe and Fairfull, 1980, 1982, 1984, 1985, 1986; Gowe and Wakely, 1954; Gowe and Johnson, 1956; Gowe et al., 1959a, 1959b, 1973; Fairfull and Gowe, 1980). Although only 40 males were used to start the control with an effective number (N_e) of parents of 132, from 1959 (the ninth generation) to 1980, each generation was pedigreed and reproduced by 80 sires and 240 dams with an N_e of 384. Each sire was mated to three dams and sufficient progeny were obtained so that a son of each sire and a daughter of each dam were used for mating the subsequent generation (Gowe et al., 1959b). By 1980, after 30 years of random breeding, the calculated inbreeding was F=6.3 percent (Gowe and Fairfull, 1985). In 1978 the actual inbreeding coefficient was F=6.1 percent which was very close (6.0 percent) to the theoretical inbreeding predicted using the formula for this type of restricted pedigree random breeding mating system (Gowe and Fairfull, 1980). Inbreeding was only increasing at the rate of about 0.13 percent per generation after the population size was increased in 1959, or 0.21 percent per generation over the 30 generations. Since 1984, 100 males and 200 females have been used to maintain Strain 5.

The value and use of Strain 5 in making it possible to separate genetic and environmental trends is illustrated in Figures 38.1 and 38.2. Egg weights were taken at

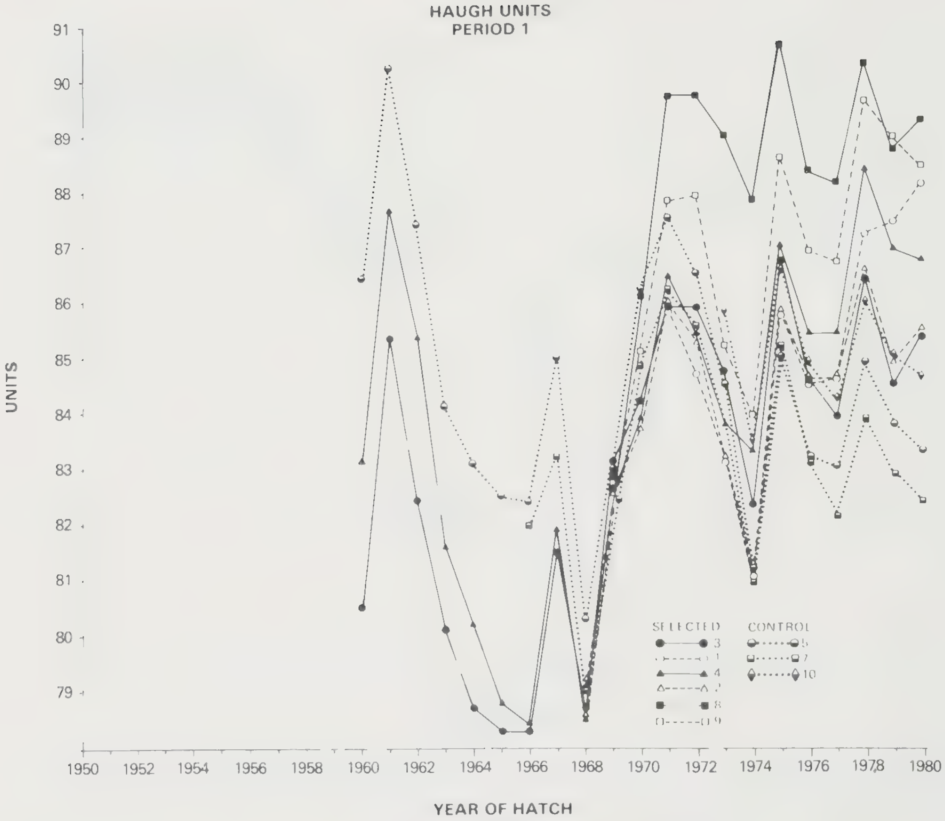

Figure 38.3. Survivor Haugh units for selected Strains 1, 3, 2, 4, 8 and 9, and unselected control Strains 5, 7 and 10. Measured at about 225 days from 1960 to 1969 and at about 240 days from 1970 to 1980.

different ages early and late in the study in which this strain was used as the control, although there was an overlap of five years when eggs were measured at both ages. The effect of age of the bird when egg size was measured was therefore confounded with generation. When the selected strains were plotted as a deviation from the control strain, both the age effect and yearly environmental effects were removed, and the genetic trend was clear. It can readily be seen that Gowe and colleagues did not select for egg size for the first few generations, then they selected for egg size using relatively ineffective pedigree data only, and finally from 1962 onward they used individual and family information and the egg size trait responded readily until selection was reduced after 1972. Without the control strain it would have been difficult to differentiate this clear genetic trend from age and environmental factors.

A new control strain started in 1958 (Strain 7, formerly called Kentville control) was introduced in 1966 into the ARC study. Another control was introduced in 1973 (Strain 10). Both of these synthetic populations were reproduced with 80 sires and 240 dams using the pedigreed random breeding restricted system (Gowe and Fairfull, 1980, 1982). This meant that it was possible in this study to evaluate whether there was any genotype x environment interaction brought about by the growing difference in mean performance

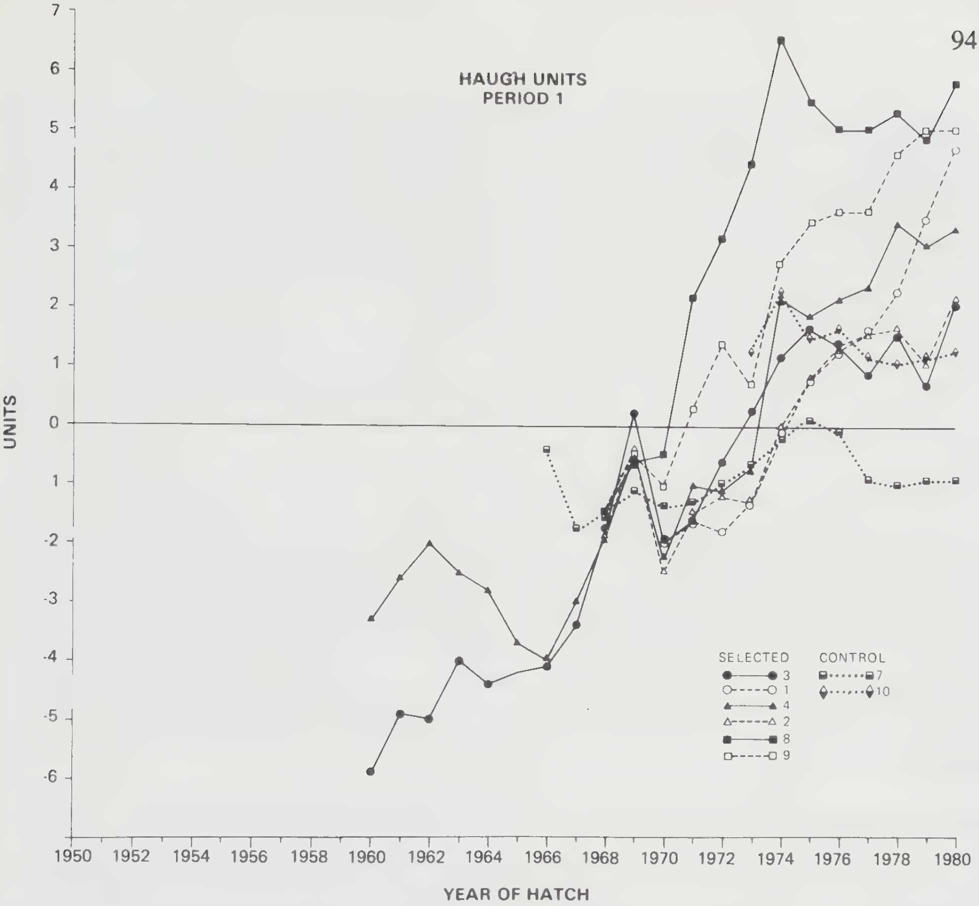

Figure 38.4. Survivor Haugh units data from Figure 38.3 expressed as deviations from the unselected control Strain 5.

between the selected strains and the original control Strain 5. For egg size it is apparent from Figures 38.1 and 38.2 that there was little evidence of a genotype x environment interaction.

Another example of the value of a control can be seen in Haugh units (albumen quality), which is very sensitive to environmental effects. The genetic trend is completely obscured by environmental fluctuations when the absolute values are plotted against generations (Figure 38.3). The genetic trend is easily seen when the selected strain means are deviated from the control strain mean (Figure 38.4).

There is no evidence that any of the three White Leghorn control strains at ARC have drifted to a degree sufficient to invalidate their use.

U.S.A.: United States Department of Agriculture (USDA), North Central Regional Poultry Laboratory (NCRPL). The Cornell randombred control population was started at Cornell University with matings in 1955 using a broad base of White Leghorn strains. The general design called for mating 50 males to 250 females, with each male contributing a male and each female a female to the next generation (King et al., 1959).

From 1956 until it was discarded, the Cornell Control Strain was maintained at the

USDA NCRPL at Purdue University, Lafayette, Indiana, with the same mating structure. It was first started as a replicate of the Cornell population and later became the sole source (King, 1961).

The Cornell or Regional Cornell Control Strain has been used in several selection studies and it has been genetically analyzed in several research papers (King et al., 1961, 1963; King, 1961; van Vleck et al., 1963; van Vleck and Doolittle, 1964; Weinland et al., 1964; Kinney et al., 1968; Kinney and Lowe, 1968).

A later study comparing the Cornell Control Strain maintained in two sizes of randombred (unpedigreed) populations with the pedigreed population, showed that the pedigreed mating structure with a sufficiently large population (N_e = 250 or more) was necessary to minimize drift effects if the control was to be used for long-term selection studies (Garwood et al., 1974). This result confirms what Gowe et al. (1959b) had suggested earlier.

In 1972, another control strain (NCR), a synthetic of six commercial Leghorn strains, was started at the USDA North Central Regional Poultry Breeding Laboratory at Purdue University (Garwood et al., 1980). The NCR control was compared to the ARC Strain 10 that was independently started the same year using four of the same six commercial stocks used by Garwood (Fairfull et al., 1983a). It was maintained as a pedigreed random breeding population with the restrictions described earlier until it also was discarded. Some physiological parameters (feed deprivation, adiposity, lymphoid leukosis and its relationship to slow feathering and other performance traits) were examined in this random breeding population (Garwood et al., 1981; Garwood and Aberle, 1985; Harris et al., 1984; Lowe and Garwood, 1982).

Denmark, Finland, Norway, Sweden: Four Country Cooperative Project. This Scandinavian control was started in Sweden from a broad genetic base using international commercial hybrids from a variety of sources. In 1971 the F_2 generation was distributed to the cooperating scientists in Sweden, Norway, Denmark and Finland (Liljedahl et al., 1973, 1979). This project was recently reviewed by Kolstad (1984). The control was subsequently maintained at research institutions in the four countries and used as a control strain and as a base population in the cooperative selection studies undertaken by scientists in these four countries (Liljedahl and Weyde, 1980; Kolstad, 1980; Sorensen et al., 1980). Genetic parameters of the base population were estimated from the F_3 generation (Liljedahl et al., 1979).

After the seventh generation there was no significant difference in the MHC haplotypes of three of the control populations maintained in Sweden, Norway and Denmark. Two years later the same result was found with samples of blood from the Finnish segment of the original Scandinavian Leghorn Control (Kolstad, 1984). There was some variation in the size of the control population maintained in each country with the minimum number of males and females used in three countries being as follows: Sweden 115 and 400, Norway 25 and 195, Denmark 50 and 227. Nevertheless, the sizes of these breeding populations are such that little genetic drift would be expected in seven to nine generations.

Kolstad (1979a, 1979b) also developed a control strain in 1969 by putting together four closed lines of Leghorns used to produce commercial stock plus a university closed flock of Leghorns. This strain was used as a control strain in the Norwegian National Random Sample Test from 1971 to 1977.

When poultry research workers at the different institutions that were known or thought to have egg stock control strains were surveyed in 1978, only the USDA randombred

(NCR), the CSIRO control, and the three Agriculture Canada (ARC) controls (5, 7 and 10) were still in existence (Gowe and Fairfull, 1979). With the publication of the Liljedahl et al. (1979) paper, it was obvious that the Scandinavian egg stock control was also in existence and it was missed in the survey. As far as the authors are aware, in 1989 only the Australian, Scandinavian, and Canadian (ARC) Leghorn control strains still existed at research institutions. With the current trend to eliminating poultry breeding research from academic and research institutions, even they may not remain for long. This is a sad commentary on the vision and understanding of senior agricultural research administrators who have not had the courage to insist that such valuable biological populations are preserved indefinitely.

Chicken Meat Stock Control Strains

Canada: AC, ARC, Ottawa. The first poultry meat control strain (Ottawa Meat Control Strain, now called Strain K) was started in 1955 by systematically combining four meat strains of chickens and random breeding this synthetic population at the ARC (Merritt and Gowe, 1962). This strain has been maintained at the ARC to the present (Chambers et al., 1981). After the four strains were combined by first making two-way crosses and then four-way crosses, the control was maintained with matings of 80 sires and 240 females using the restricted pedigree system (Merritt and Gowe, 1962). Since the Ottawa Meat Control represented the genetic stock available to producers in the mid-1950s, it has been a valuable strain in assessing the effects that modern meat selection programs have had on growth, conformation, body fat, and composition (Chambers et al., 1981).

Merritt (1966, 1968) has reported on the genetic parameters of this early meat control strain. A random sample of the original strain was sent to the USDA Regional Research Station at Athens, Georgia in 1958 and maintained independently at that location (Hess, 1962). It was named the Athens-Canadian Randombred Control. By exchanging samples of the Ottawa Control, comparisons were made on the performance of the two segments. There was little difference in the performance of the USDA segment and the Ottawa-based population when tested at Ottawa over two years (Merritt and Gowe, 1962).

When the Athens-Canadian meat control was tested at two locations in the southern United States, there were some minor changes in performance relative to the Ottawa Meat Control over time. Changes were mostly nonsignificant except for a significant decline in adult body weight at one location and a significant increase in egg production at another location (Marks and Siegel, 1971). These changes were later attributed to the environment (Marks, 1971b). When the Ottawa Meat Control from ARC and the Athens-Canadian randombred from the USDA Regional Laboratory were compared at Athens over seven years there was no evidence of a divergence in the two populations, suggesting that both were genetically stable (Marks, 1971b). At both locations there was a change in the gene frequency for comb types, probably associated with natural selection against rose comb which is known to be associated with low fertility (Crawford and Merritt, 1963).

Five sire line stocks were obtained from commercial poultry meat breeders in 1978 to form a new random breeding sire strain control (ARC Strain 20) at the ARC. By first making two-way crosses, and then four-way crosses, followed by random breeding using the pedigree system with restrictions, a new base population was formed that was reasonably typical of 1978 commercial meat sire stocks. About 60 males and 180 females are used each generation to reproduce this meat control (Chambers et al., 1984). The performance of this control (ARC Strain 20) and three selected strains derived from this base population over five generations were described by Chambers (1987).

At the same time the sire control strain was formed, a dam control strain (ARC Strain 30) was started from seven commercial broiler dam stocks obtained in 1978. It was established in the same way as the sire control strain, with three generations of crossing, and then random breeding using the restricted pedigree procedure. Selected strains were derived after one generation of random breeding (Chambers et al., 1984). This strain and two selected strains derived from it were used to establish genetic parameters for growth, fatness, feed efficiency, and also the level of very low density lipoproteins (VLDL) in the blood.

U.S.A.: USDA, Southern Regional Poultry Laboratory (SRPL), Athens. The Athens Randombred (ARB) strain was started in 1958 at the SRPL, Athens, Georgia. The males used came from eight commercial broiler stocks, and the females came from eight experimental strains of different breeds, most of which were dual-purpose (Hess, 1962). This strain was evaluated later by Marks (1971a). The sex-linked dwarf gene (*dw*) was introduced into both the Athens-Canadian and the Athens meat control strains by repeated backcrossing to examine the expression of the *dw* gene in these two stabilized control strains (Marks, 1981). This research confirmed that the heterogametic Dw^{+}/dw male grew at a slightly slower rate than the Dw^{+}/Dw^{+} male and this effect increased as the birds matured.

Turkey Control Strains

U.S.A.: Ohio State University. The institution that has made the biggest contribution to turkey breeding knowledge, including control strains, is the Poultry Science Department of Ohio State University, Wooster, Ohio.

Dr. M.G. McCartney started the first Ohio Randombred Control (later called RBC1) in 1956 by crossing four strains of White Holland turkeys. These four strains were made into a synthetic by first crossing them in all possible combinations and subsequently random breeding without selection. In the early years, 48 sires and 96 dams were used in a restricted pedigree program. Later the control was maintained with 48 pair matings (McCartney, 1961, 1962, 1964). Selected strains for egg production and body weight were derived from this control (Nestor, 1980; Nestor et al., 1967; McCartney et al., 1968).

A second randombred control (RBC2) was started at Wooster in 1966 from reciprocal crosses of two commercial 'elite' strains (Nestor, 1977a; Nestor et al., 1969). The two Ohio controls, RBC1 and RBC2, were compared over nine generations, and appeared to be relatively stable, with only 36 pairs mated in each strain each generation (Nestor, 1977b). Genetic parameters of the RBC2 control were reported for body weight, skeletal characteristics, and carcass composition by Havenstein et al. (1988b, 1988c) and Nestor et al. (1987, 1988).

Japanese Quail Control Strains

U.S.A.: Ohio State University. Several institutions have maintained randombred control populations of Japanese quail for various kinds of genetics and physiology research. However, little research has been undertaken with this species on the design and efficiency of various types of control. Recently, Havenstein et al. (1988a) compared the use of pedigreed and unpedigreed randombred control systems for selection studies in quail. They compared three pedigreed and three nonpedigreed lines each with 32 pair matings. With this species, where pair matings in cages are the easiest way to manage these birds, the authors concluded that the nonpedigreed mating system was as efficient as the pedigreed system. However, because of the well known susceptibility of Japanese quail to inbreeding, they recommended a larger number of pair matings than the 32 pairs that they used, particularly when the control is to be used with long-term selection experiments. Earlier, Nestor et al. (1982) reported on a selection study with quail at Ohio State University in which a randombred control was maintained.

Other quail control strains. Other institutions have reported on the maintenance and use of Japanese quail randombred control populations. Researchers at Washington State University maintained a large unpedigreed random breeding population of quail, with 800 females and 160 males, which was large enough to be relatively stable even though unpedigreed (Macha and Becker, 1976).

Marks (1978) at SRPL has reported on a long-term selection study for growth in quail in which he used a control strain for 40 generations. Garwood and Diehl (1987) combined the Ohio State University quail control with the SRPL control to form a broad-based quail control for their selection studies.

Waterfowl Control Strains

There are remarkably few selection studies or other genetic research with waterfowl where control strains have been reported, and even when reported, the details are sketchy. Powell (1984) reported on a selection program of a commercial duck breeding company in Great Britain, where a control was maintained for comparison against lines selected for feed efficiency. He noted that there was some mild selection pressure for growth in the control population. Pingel and Heimpold (1983) and Pingel et al. (1984) also used a control in their duck selection studies on feed efficiency.

The Animal Research Centre at Ottawa has been maintaining two goose random breeding control populations for several years that are used in their selection and management studies with geese (CCAC, 1985).

CONCLUDING REMARKS

There is no doubt that some adequate way to separate genetic and environmental trends in both academic breeding studies and commercial breeding work is necessary. In short-term single trait academic selection studies the best alternative is a pedigreed random breeding control of an adequate size. The size will depend upon the length of the study and the type of traits being studied. For a trait with a high standard deviation, such as egg production, the size will need to be larger than for a trait such as growth. This control should originate from the same base population as the selected line(s). If the base population is a highly selected strain or a newly formed synthetic (composite), it is essential that several generations (three would be adequate) of random breeding precede the start of the selection experiment.

A study by Foster and Thompson (1980) on the genetic drift in three small populations over nine generations illustrated pitfalls to be avoided when developing control strains. The main deficiency is that they crossed two distinct strains and started the study without waiting for the stocks to come into equilibrium. Epistatic combinations were likely still breaking up during the experimental period. Furthermore, this study demonstrated that populations with an N_e varying from 27 to 40 are much too small to minimize drift, confirming earlier theoretical and empirical studies (Gowe et al., 1959a; Garwood et al., 1974). In long-term experiments and in industrial breeding programs, larger control breeding populations with an effective number of 300 or more should be used whenever possible.

While inadequate controls are not satisfactory, controls with a sufficiently large N_e appear to be stable in practice. Controls held at different sites (Merritt and Gowe, 1962; Marks, 1971b; Kolstad 1984), or the relative performance of different controls over generations (Nestor, 1977b), have been compared and found to be phenotypically stable. The controls used in the Ottawa long-term selection study were relatively stable and served to remove the environmental trends from the results. This can be seen in Figures 38.1 to 38.4 and in Gowe and Fairfull (1985, 1986).

Commercial breeders need controls at two levels in their programs: as a control on genetic change in their pure lines, and as a control on the change in performance of their commercial (strain cross) products as well as that of their competitors. When making comparisons of commercial products, the preferable control would be a strain cross of two stabilized control populations already in equilibrium that were known to combine well. Therefore, the control would be expressing a high level of heterosis (hybrid vigor) like the commercial products.

Flock (1988) has recently discussed some of the practical problems facing those developing suitable controls. If randombred pedigreed control populations are kept large enough to minimize drift and inbreeding, and crosses of stabilized control lines are used when testing commercial strain crosses, most of Flock's concerns would not be important. A major permanent change in the environment in the course of an experiment, such as the shift of adult birds from floor pens to cages about halfway through the selection study reported by Gowe and Fairfull (1985), had a large permanent effect on full-year performance of the control line. The presence of the control made it possible to identify and quantify this interaction of genotype and environment, and to explore its biological basis. The analysis and interpretation of events is of critical importance in research and applied breeding. Control populations are an integral part of good research and development work, and should not be neglected by poultry breeders and research scientists.

If the only use of pedigreed randombred control strains were retrospective studies, it would still be very worthwhile to maintain them. For example, controls were used to assess changes in body composition and growth and biochemical parameters as a result of selection in broilers (Chambers et al., 1981; Grunder et al., 1987). The relationship between MHC haplotypes and Marek's disease resistance was made clearer by a study involving changes in haplotype frequency and Marek's disease resistance in selected and control strains (Gavora et al., 1986). With the growing importance of molecular genetics in poultry breeding, the value and the need for maintaining large stable random breeding base populations in selection experiments will become increasingly important. When the Ottawa long-term selected strains and their controls were compared for their frequency of endogenous leukosis genes, it was clear that selection for high egg production and associated traits had eliminated some endogenous genes and reduced the frequency of others (Kuhnlein et al., 1989). Without large stable control strains, it would be much more difficult to carry out such studies.

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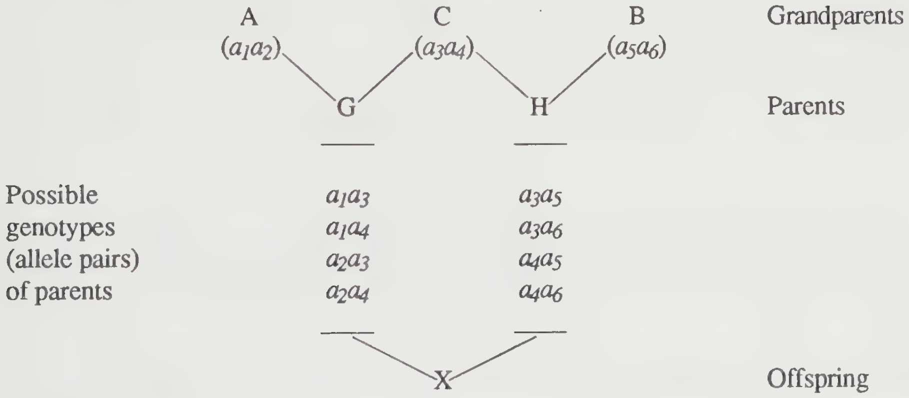
Chapter 39. INBREEDING

Hans Abplanalp

THE CONCEPT OF INBREEDING

Inbreeding results from mating of relatives. Two individuals are genetically related when they have common ancestors from which genes have come to them. The degree of inbreeding of an individual depends on how closely related its parents were; that is, on how many ancestors are in common and how often and how recently they appear in the pedigree, the list of ancestors. The coefficient of inbreeding (F) serves as a measure of inbreeding and can be defined as the probability that an individual in question carries, at any given locus, two genes that are derived from an identical ancestral gene (identity by descent). For example, the following will describe the calculation of F for an individual X resulting from a half-sib mating where the two parents (G and H) have a common sire (C) but two different dams (A) and (B). The six ancestral alleles carried in the three ancestors for a typical locus (A) are designated by subscripts as follows:



Among the possible genotypes of parent G the allele a_3 appears twice; similarly there are two ways in which H can carry that gene; so when mated together G and H can potentially produce homozygous a_3a_3 progeny in four different ways. Similarly allele a_4a_4 can be produced with the same frequency among a total of 64 allele combinations in progenies from a G x H mating. Thus the probability of either a_3a_3 or a_4a_4 genotypes among potential progeny is

$$P(a_3a_3) + P(a_4a_4) = 4/64 + 4/64 = 1/8 = F_X$$

This probability is the inbreeding coefficient, (F_X) of individual X, which is thus defined as the probability that the two alleles carried by X at a typical locus are derived from a single allele carried by their common ancestor (C). This definition must then be extended to include all possible common ancestors in the known pedigree of X. The same reasoning applies to all genetic loci. Thus, inbreeding (F) is defined regardless of the number of ancestral genes under consideration. However, if a common ancestor is itself inbred with respect to still more remote ancestors (A), inbreeding (F_A) must be taken into account, as will be shown later. Since exact inbreeding calculations take only pedigree information into account it must be assumed that at some remote ancestral generation inbreeding (F_A) was either zero, or of a predetermined level, based on nonpedigree information.

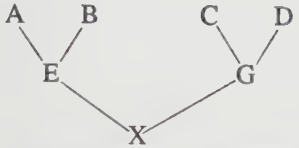
Because the coefficient of inbreeding (F_X) is a probability, it says nothing about the precise genotype of X. Thus genes homozygous in ancestors for unknown reasons are homozygous in state, but do not contribute to inbreeding as calculated from the pedigree. Also, brothers and sisters of X have the same pedigree; their inbreeding coefficients are all identical even though individuals actually vary in their genotypes.

Poultry geneticists were among the first to recognize the need for a systematic evaluation and definition of the degree of inbreeding. Working with close matings such as parents mated to offspring (Dunkerly, 1930), or brothers mated to sisters (Goodale, 1926, 1927; Dunn, 1923; Jull, 1929a, 1929b), it was sufficient in these early studies to state the numbers of successive generations that a given regular system of mating had been employed. A more general numerical estimate of inbreeding, applicable to any breeding system was first devised by Raymond Pearl (1913). However, his derivation of inbreeding levels was based on erroneous assumptions and is unusable. His work, however, became known to Sewall Wright (Crow and Dove, 1987) who obtained a general solution to the calculation of inbreeding coefficients, first for regular systems of mating (Wright, 1921), and later for the general case of individuals with known pedigrees (Wright, 1922), leading to his well known inbreeding formula:

$$F_X = \frac{1}{2} \sum_i {\frac{1}{2}}^{n_i + m_i} (1 + F_{A_i})$$

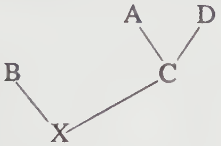
In the above formula each common ancestor (A_i) must be considered and all the possible ways in which its genes might be passed to both parents of individual X. For each such case the number of reproductive steps between the common ancestor (A_i) and the male parent (n_i) is added to the number of steps between A_i and the female parent (m_i) and their sum used as exponent of 1/2. Also taken into account is the inbreeding coefficient of the common ancestor, $F(A_i)$, under consideration. Worked examples for an application of Wright's formula to irregular pedigrees have been given by Falconer (1981), Pirchner (1983), and Lerner (1950) among others. An important feature of these calculations is their rapidly mounting complexity as ancestral generations are added to a pedigree, and especially in small flocks where the same individual may appear in numerous ways as a common ancestor.

Wright's method of inbreeding computation becomes unwieldly in pedigrees with many generations and close mating systems. More manageable procedures based on the genetic relationship or coancestry potential of mates have been developed by Emik and Terrill (1949) and by Cruden (1949). The coefficient of coancestry used by Cruden (1949) equals the coefficient of inbreeding of potential progeny as shown in the diagram below; thus $f_{EG} = F_X$. The coancestry of two mates then can be computed from the average coancestries of four pairs of hypothetical matings between sets of parents.



$$f_{EG} = (f_{AC} + f_{AD} + f_{BC} + f_{BD})/4 = F_X$$

In this manner one generation's coancestries can be computed from coancestries of the preceding generation. Cruden's (1949) method is applicable where generations do not overlap. For overlapping generations, Emik and Terrill's (1949) method applies. It averages coancestries of a given individual with the parents of its mate:



$$f_{BC} = (f_{BA} + f_{BD})/2 = F_X$$

This latter method can be used under all circumstances as demonstrated in Appendix A.

Regular Systems Of Inbreeding

Wright's (1921) pioneering study of regular mating systems was not based on the probabilistic concepts given above but on an analysis of correlations and regressions among numerically assessed genes passed on to a given individual. Wright (1922) thus defined the coefficient of inbreeding as the correlation of uniting gametes within the context of a randomly mating population. Pirchner (1983) gave a direct derivation of this definition of F.

In regular mating systems of close relatives, such as full-sib matings, matings of offspring with the younger parent, or that of double first cousins, the inbreeding coefficient rises steadily with each generation until it reaches its theoretical value of 1.0 or 100 percent (Table 39.1). At that point the line is completely inbred and theoretically homozygous at all loci.

Inbreeding (F) can be brought into relation to heterozygosity (H) of a given locus with respect to its initial heterozygosity (H_O) in a noninbred base population. Thus in generation t:

$$H_t = H_O(1 - F_t) \text{ and } F_t = (H_O - H_t)/H_O$$

showing that F_t at any generation equals the loss in heterozygosity relative to that present in the noninbred base flock ($t = 0$). From these relations it follows that for a completely inbred line ($F = 1$ or 100 percent) all loci should be homozygous for one or the other allele. Such inbred lines are called isogenic. Since it is uncertain which alleles are homozygous or fixed, it follows that numerous isogenic lines derived from the same source population differ in their genetic makeup.

The rate ΔF at which inbreeding progresses per generation under a given mating system is defined as:

$$\Delta F = \frac{F_t - F_{t-1}}{1 - F_{t-1}} = \frac{H_{t-1} - H_t}{H_{t-1}}$$

In regular mating systems, such as full-sib mating, the rate of inbreeding initially progresses somewhat irregularly, but eventually settles down to a constant limiting rate as shown in Table 39.1. It can also be seen that the rate of inbreeding is inversely related to the number of parents reproducing each line. Thus for full-sib matings with just two individuals mated (N=2), the limiting rate of inbreeding is 19 percent as shown in Table 39.1. For half-sib matings with one male and two females (N=3) ΔF=13 percent, and for double first cousin matings (N=4) ΔF = 8 percent. Continued mating of offspring with the younger parent (N=2) results in the same rate of inbreeding as that of full-sibs.

The last mating system in Table 39.1, that of repeated backcrossing of the most recent offspring to a single ancestor, does not result in an isogenic line if the ancestor is itself noninbred. The limiting inbreeding level attained is then 50 percent. The reason for this is easily seen by considering a locus that is heterozygous (a_1a_2) in the common ancestor. In this case homozygosity present in mated offspring (a_1a_1 or a_2a_2) would be preserved in only half of the next generation's individuals. Ultimately progeny from repeated backcrossing to a single ancestor (A) nevertheless carry 100 percent of its genes, which is equivalent to one generation of hypothetical selfing of A.

Inbreeding In Larger Populations

From mating plans with close regular inbreeding, the inverse relationship of population size and the rate of inbreeding can readily be seen (Table 39.1). It is also clear

Table 39.1. Coefficients of inbreeding (F) in successive generations for some regular systems of close matings.

Generation	Mating system[1]				
t	A	B	C	D	E
1	0	0	0	0	0
2	0.250	0.125	0.125	0	0.250
3	0.375	0.250	0.219	0.125	0.375
4	0.500	0.344	0.305	0.188	0.438
5	0.594	0.430	0.381	0.250	0.469
10	0.895	0.716	0.691	0.508	0.499
$\Delta F = \frac{F_t - F_{t-1}}{1 - F_{t-1}}$	0.19	0.13	0.11	0.08	0.00

[1] Mating system:

A: Full-sib or parent-offspring (N = 2).
B: Half-sib: One male to two half-sisters which are full-sisters of each other (see Figure 39.1) (N = 3).
C: Single male mated to large number of half-sisters.
D: Double first cousins (N = 4).
E: Backcrosses to a constant parent (N = 2).

that common ancestry, the cause of inbreeding, becomes unavoidable in flocks of any size. Thus under full-sib mating (N=2), the mates unavoidably have both parents in common. Under double first cousin matings (N=4), common ancestors become unavoidable in the grandparent generation; even with flocks of 512 pairs (N=1024), common ancestry would become unavoidable after ten generations since the number of independent ancestors would then have to be 2^{10} or about 1000.

The rate of inbreeding in a flock of size N with random pair mating of N/2 males with N/2 females is expected to be $\Delta F = 1/2N$.

In randomly breeding flocks with Nm sires mated to a total of Nf dams, inbreeding rises at a rate of:

$$\Delta F = 1/8Nm + 1/8Nf$$

Thus for a flock of six sires each mated to five dams the relative increase of inbreeding per generation is:

$$\Delta F = 1/(48) + 1/(240) = 1/40 \text{ or } 2.5 \text{ percent}$$

Using the rate of inbreeding as a measure of flock size, one can equate an observed rate of inbreeding to that expected for an idealized pair-mated population and call that number the effective size (Ne) of the flock in question. Thus, in the above case $\Delta F = 1/40 = 1/2Ne$, giving an effective size of Ne = 20.

From considerations of common ancestry and the definition of the inbreeding coefficient, it follows that inbreeding in a flock is cumulative and its increase is linear over the short-term, especially if populations are large. The level of inbreeding, once attained, cannot be reduced by special mating schemes unless the flock in question had previously been subdivided into sublines. However, it is possible to control the rate of inbreeding in a population by ensuring that each male leaves one son and each hen exactly one daughter. This restricted reproduction will double the effective population size (Ner) under pair mating to:

$$Ner = 2Ne$$

For a flock with Nm sires and Nf dams, Gowe et al. (1959) have shown effective population size under restriction to become:

$$1/Ner = 3/16Nm + 1/16Nf$$

In the above case each male is mated to several dams, and the number of sires (Nm) will dominate the rate of inbreeding; under restricted family size the number of sires (Nm) becomes even more important. Applications of the above principle in the case of randomly selected control populations have been considered by Gowe et al. (1959) where savings in bird numbers are of considerable importance.

A second widely applied practice is the avoidance of matings between full-sibs, half-sibs and possibly other close relatives. Avoiding full-sib matings in a pair-mated flock retards ΔF only slightly from random mating to:

$$Ne = N + 1.5 \text{ or } \Delta F = 1/2(N + 1.5)$$

When compared with the restriction of progeny to one per parent, this avoidance of close matings is much less effective.

The combined effects of these two procedures on F have been worked out by Robinson and Bray (1965) who confirmed the above conclusion.

The avoidance of close matings in breeding flocks under artificial selection makes good sense, because the resulting inbred families would be expected to perform below the level of noninbreds and hence be less likely to leave selected offspring, even if their additive breeding values were equal.

Artificial selection for highly heritable traits among progeny, or the use of family selection, tend to reduce effective population size (Ne) substantially below that expected from the number of mated parents. Artificial selection thus not only affects the characters under selection but increases the rate of inbreeding above that of a randomly selected flock of the same size. These effects can be taken into account in pedigreed populations for which inbreeding coefficients are calculated each generation. The rate of inbreeding (ΔF) can then be used to estimate effective population size as shown above.

An example of the effects of artificial selection in a number of small commercial flocks of turkeys is shown in Table 39.2 based on results of Cahaner et al. (1980). Estimated population sizes (Ne) based on the number of mated sires (Nm) and dams (Nf) are seen to be substantially larger than those based on ΔF between the 1977 and 1978 generations of progeny. These estimates, while subject to considerable variability, reflect the effects of artificial selection for reproduction and growth rate. Based on the average of eight lines shown in Table 39.2, effective population size estimated from inbreeding changes was Nef=26 vs. Ne=35 estimated from the number of mated parents.

Estimates of effective population size based on the rate of inbreeding require inbreeding calculations over several generations in order to arrive at stable ΔF values. If long-term pedigrees are not available, effective population size can be estimated on the basis of variability in family sizes. Such estimates are based on the concept of the sampling

Table 39.2. Estimates of effective flock size (N_e) from numbers of reproducing sires (N_m) and dams (N_f), and from the increase in average inbreeding (N_{ef}), for eight turkey flocks under selection for reproduction and growth. Based on data from Cahaner et al. (1980).

Line	1977 matings		Average inbreeding (%F)		ΔF[1] x 100	Effective flock size		
	Sires N_m	Dams N_f	1977	1978	1978-77	N_e[2]	N_{ef}[3]	N_{ef}/N_e
1	12	33	13.58	14.44	1.00	35.2	50.0	1.42
2	13	33	7.22	10.19	3.20	37.3	15.6	0.42
3	11	37	7.66	9.34	1.82	33.9	29.8	0.88
4	11	36	10.08	11.87	1.99	33.7	25.1	0.75
5	12	33	7.38	9.54	2.33	35.2	21.5	0.61
6	12	42	5.45	7.21	1.87	37.3	26.7	0.72
7	12	44	7.97	9.39	1.53	37.7	32.7	0.87
8	10	36	6.28	9.00	2.90	31.3	17.2	0.55

[1] $\Delta F = (F_{78} - F_{77})/(1 - F_{77})$

[2] $N_e = 1/(1/4N_m + 1/4N_f)$

[3] $N_{ef} = 1/(2\Delta F)$

variance of gene frequencies (V_q) expected in the reproduction of mated parents.

Since a population of N individuals represents 2N genes passed on to them from the previous generation, the binomial variance in their gene frequency would be expected to be approximately $\frac{q(1-q)}{2N}$ under pair mating. This also is the variance in the change in gene frequency between generations; thus:

$$Vq = \frac{pq}{2N} = Fpq$$

However, these relations are modified in a real-world situation by genetic variability in the reproduction of mated parents. Thus if the variance in family size (Vn) is known, the effective size of a population can be expressed in terms of a randomly reproducing pair-mated population. This variance effective size (Nev) has been shown to be:

$$Nev = \frac{4N}{2 + Vn}$$

with a total of N parents each generation. In a randomly reproducing stable population the variance in family size (V_n) is approximately 2, giving an effective population size (Nev) equal to N.

In poultry breeding, matings are usually hierarchical with one male mated to several females. Under such hierarchical matings variability in family size must be determined for four equally important ways in which genes pass from parent to offspring, namely: sire to son (MM), sire to daughter (MF), dam to son (FM), and dam to daughter (FF). Corresponding family sizes (n_{mm}, n_{mf}, n_{fm} and n_{ff}) and variances in family sizes can then be considered (refer to Appendix B).

For constant population size and structure Hill (1972) thus showed effective population size to be:

$$\frac{1}{Ne} = \frac{1}{16N_m}\left[2 + Vmm + \frac{2N_m}{N_f} Cov\,(mm.mf) + (N_m/N_f)^2 V_{mf}\right]$$

$$+ \frac{1}{16N_f}\left[2 + (N_f/N_m)^2 V_{fm} + \frac{2N_f}{N_m} Cov(fm.ff) + V_{ff}\right]$$

Thus, if variances of family sizes can be estimated from pedigreed offspring, effective population size (Ne) can be assessed, and from it the expected rate of inbreeding (ΔF). A numerical example of estimation of effective population size based on variances of family sizes is given in Appendix B.

With six sires each mated to five dams the expected effective flock size is 20 under random reproduction. However, artificial selection of the parents from a larger flock of available males and females leads to a reduction in effective size to Nev = 9.046.

It should be noted that Hill's (1972) formula assumes a constant population size and structure. If the population expands or contracts in numbers of selected parents, the more general formulas given in Table 39.14 must be applied in accordance with derivations by Latter (1959). In the case of poultry flocks with nonoverlapping generations, a calculation of variances in family size must be made to reflect a representative stage in the flock's history, such as the time when male parents are placed in breeding pens as shown in the example of Table 39.13.

Hill's (1972) formula can be used to derive effective population size under random mating on the assumption that numbers of reproducing family members follow a Poisson distribution where the mean equals the variance. Under these conditions and stable population structure:

$$\begin{aligned} V_{mm} &= V_{ff} = 1 \\ V_{fm} &= N_f/N_m \\ \text{and cov (mm, mf)} &= \text{cov (fm, ff)} = 0 \\ \text{thus } 1/N_e &= 1/4N_m + 1/4N_f \end{aligned}$$

The impact of mass selection on effective population size has been shown by Robertson (1961) to depend on the heritability (h^2) of the selection criterion as well as the selection intensity (i). Thus, for a population with pair matings, the ratio of actual to effective flock size is:

$$Ne/N = \frac{1}{1 + 2i^2 h^2}$$

Effective population size is still further reduced if family selection is applied. In such a situation an empirical determination of effective population size from family variances remains the most appropriate method of assessment. With mass selection of moderate intensity for a highly heritable trait such as body weight, a reduction of effective population size to half that expected from random mating is not uncommon, as shown in the examples of Table 39.2 and Appendix B.

Genetic variances under inbreeding. Under inbreeding in reasonably large flocks, within-line variance (V_A) is expected to decline in proportion to (1 - F); thus:

$$V_{At} = V_{Ao} (1 - F)$$

In small lines, such as with full-sib matings, the within-line variance starts out initially below the level of the base population from which they are being derived because lines originate from specific full-sib families within that line. Thus $V_{At} = V_{Ao} (1 + F_t - 2F_{t+1})$ where F_{t+1} is the inbreeding of the mated birds' potential offspring. Thus, in a randomly mating base population the variance within families of full sibs is $V_{Ao} [1 + 0 + 2 (0.25)] = 0.5 V_{Ao}$.

Genetic variance between inbred lines derived simultaneously from the same base population (V_{Bt}) increases in proportion to F for additively acting genes. Thus:

$$V_{Bt} = V_o(2F_{t+1})$$

Numerical values for within and between line genetic variances under continued full-sib

Table 39.3. Expected genetic variances within and between inbred lines under full-sib mating. Values shown are in relation to a total genetic variance V_{ao} of 1.00 in the base population from which all lines are derived.

Generations of sib mating	F	Additive genes		Recessive genes q_o (aa) = 0.1[1]		Recessive genes q_o (aa) = 0.02	
(t)	%	V_w	V_b	V_w	V_b	V_w	V_b
0	0	0.50	0.50	0.700	0.300	0.740	0.260
1	25	0.50	0.75	2.087	1.057	9.625	3.563
2	38	0.38	1.00	2.040	2.143	10.183	9.043
3	50	0.31	1.18	1.973	3.224	10.263	14.978
4	59	0.25	1.34	1.717	4.225	9.104	20.628
5	67	0.21	1.46	1.463	5.088	7.843	25.062
10	89	0.01	1.98	0.542	7.628	2.945	40.690

[1] q_o = frequency of recessive gene in base population

mating are given in Table 39.3.

In very small populations, such as full-sib mating lines, inbreeding may lead to an initial increase in the within-line genetic variances for traits controlled by recessive genes as shown by Robertson (1952). This increase of intra-line variance occurs only for rare recessive genes which are almost completely hidden in outbred flocks (homozygous recessives at frequency q^2) but which may appear at a greatly increased frequency ($q^2 + qF$) within inbred lines. Intra-line genetic variance due to recessive genes reaches a maximum at inbreeding levels of 50 percent and then declines under further inbreeding as lines become completely isogenic. Between-line genetic variances under the recessive gene model increase over the entire range of inbreeding. Table 39.3 shows variances under continued full-sib matings for initial frequencies of recessives of $q_o = 0.1$ and 0.02.

Under the above recessive gene model the newly displayed genetic variance has both additive and dominance components. Applied to traits which show inbreeding depression, this means that selection against homozygous recessives can be effective and result in a reduced level of depression. Maximum effectiveness of such selection against recessive genes is expected at inbreeding levels of 50 percent.

It is noteworthy that between-line variances due to rare recessive genes can increase relatively more than the variances caused by additive genes. However, experimental verification of such predictions remains problematic in poultry, where inbreeding depression in reproduction leads to the selective elimination of lines with low reproductive capacity and viability. The surviving lines would then underestimate genetic variances. Unpublished results from the author's experiments of full-sib inbreeding generally conform to the expectation of increased within-line variances for egg production up to F = 50 percent, followed by a decline under higher levels of inbreeding (Table 39.4). Between-line variances also show an increase up to F = 50 percent but decline thereafter as a probable consequence of the loss of lines with severe inbreeding depression.

Table 39.4. Variance components (V) and degrees of freedom (Df) within and between lines of White Leghorn hens inbred by full-sib mating for survivor egg production to 50 weeks of age. (Author's data).

				Within Lines			
Generations of	F	Between lines		Full-sib families		Individuals/families	
sib mating	%	V	Df	V	Df	V	Df
0 (base line)	0	–	–	330	514	880	704
1	25	409	114	–	–	921	311
2	38	216	62	–	–	945	226
3	50	143	33	–	–	1006	123
4-8[1]	69-83	248	63	163	90	568	481
9-13	86-94	623	33	62	101	578	259

[1] Pooled data from several generations are based on lines expanded from single pair matings to several pairs per line.

INBREEDING DEPRESSION

In all domestic bird species studied to date inbreeding has been shown to cause a decline in traits affecting reproduction and viability. Systematic investigations of inbreeding effects were carried out by Pearl (1913), Dunn (1923), Jull (1929a, 1929b), Goodale (1926), and Cole and Halpin (1922) to study the feasibility of developing highly inbred lines of chickens by continued full-sib matings. In doing so they attempted to duplicate successful breeding systems employed for commercial crosses in plants.

Experiments extending over three or more generations of full-sib mating soon demonstrated that inbreeding had pronounced effects on traits important to the propagation of lines and to their use in commercial applications. The traits most severely affected were hatchability of fertile eggs, viability of young birds, and egg production of surviving hens. Table 39.5 shows hatchability for several of these studies. In all cases but one the percentage of fertile eggs that hatched under sib mating dropped to a third to a half of that present in noninbred base populations. This situation led Waters and Lambert (1936) to conclude that early attempts to inbreed poultry resulted almost universally in disaster. Only their own experiments led to the permanent establishment of highly inbred lines. They attributed their success to strong selection for good hatchability among a large number of potential breeding hens from which the best families were selected to reproduce each line. In one line full-sib mating was used successfully for eight consecutive generations. Thus, hatchability of fertile eggs fluctuated between 80 and 100 percent over generations, ending at 81 percent in the tenth generation. Similarly egg production and fertility remained almost unchanged, but age at first egg and egg weight declined.

From the less successful studies of Jull (1929a, 1929b) and others, it was concluded that the first generation of full-sib or half-sib mating produced the most drastic losses in reproductive performance.

A number of investigations of inbreeding depression were initiated at the author's laboratory to separate its presumed causes experimentally. The factors under consideration

Table 39.5. The effects of continued brother x sister mating on hatchability of fertile eggs in early experiments with chickens.

		Generations of full-sib mating (F)			
Reference	Breed	0	1(25)	2(38)	3(50)
Cole and Halpin (1922)	Rhode Island Red	67.0	49.0	41.0	18.0
Dunn (1923)	White Leghorn	76.8	49.2	42.0	21.7
Jull (1929a)[1]	Barred Plymouth Rock	76.2	42.6	44.2	23.2
Jull (1929b)[1]	White Leghorn	81.0	62.5	46.3	43.3
Waters and Lambert (1936)	White Leghorn	90.0	61.0[2]	82.0	93.0

[1] Includes few half-sib matings in single pens.
[2] Average of two generations with F=35 percent.

were effects of directional dominance expressed in an increased frequency of harmful homozygous genotypes under inbreeding, effects and efficacy of natural and artificial selection against inferior homozygotes, and the presence of epistatic gene effects responsible for nonlinear declines of performance under inbreeding.

In a first series of experiments several poultry species were investigated by means of three generations of successive full-sib mating (Abplanalp, 1974; Sittmann et al., 1966). The first generation of these was set up with special care to produce offspring from sib-mated pairs as well as nonsib matings from the same outbred base flock, thus minimizing possible effects of selection. A second experimental generation was then produced in which crosses of inbred unrelated birds were included. The latter produced noninbred progeny from inbred dams, thus allowing estimates of inbreeding effects of maternal traits. Selection among inbreds could be assessed by comparing such crosses to control matings from the base population. Second and third generations of full-sib matings were produced without favoring families with good performance, leading to the eventual loss of most lines. Crosses among unrelated advanced generation inbreds served for an assessment of selection in preceding generations.

Table 39.6 shows the results of four studies including White Leghorns (Abplanalp, 1974), Broad-breasted Bronze turkeys (Abplanalp and Woodard, 1967), Japanese quail (Sittmann et al., 1966) and red-legged partridges (Woodard et al., 1982). In all cases inbreeding levels from full-sib matings were 25 percent relative to the base population from which they were derived and the performance for each trait of inbreds is expressed as a percentage of noninbreds in the base population. Hatchability of fertile eggs, egg production, and viability all suffered inbreeding depression. Fertility was reduced only in Japanese quail and partridges, which were naturally mated in cages. For turkeys and chickens, artificial insemination was used including substitutions for nonsemen-producing males before the onset of hatches. Since sperm cells function as haploids, little inbreeding depression is expected for their fertilizing capacity as such, and none was observed with artificial insemination.

In the case of hatchability, crosses of inbred mothers to unrelated inbred males allowed an assessment of inbreeding depression in maternal characteristics. These estimates are given separately in Table 39.6 and show that maternal inbreeding influenced hatchability, but to a lesser extent than inbreeding of the embryo. Thus in chickens a loss of about nine percent in hatchability resulted from embryo inbreeding, while hens' inbreeding reduced it by only three percent. In turkeys inbreeding effects on hatchability were more pronounced than in chickens, with substantial maternal effects (eight percent). Even more pronounced inbreeding effects were observed for Japanese quail and partridges, with inbreeding depression of almost 30 percent for embryo viability and 14 percent for maternal inbreeding.

Viability of hens showed substantial inbreeding effects, with Japanese quail again having the most pronounced depression of almost 20 percent, and chickens least with about five percent. By multiplying the probabilities of survival and reproduction of all contributing traits, total reproductive fitness could be calculated; it shows a relative survival probability of inbred hens to mating age of 74.4 percent for chickens, 61.6 percent for turkeys, 35.9 percent for Japanese quail and 34.1 percent for partridges.

Inbreeding had only slightly adverse effects on body weight of hens, but would undoubtedly be of practical importance in meat producing birds. Egg size and age at first egg showed slight, if any, effects from inbreeding. It should also be noted that maternal inbreeding was only important for hatchability, but relevant results are not shown for other traits.

The above results show pronounced species differences, and it is tempting to consider their genetic basis.

Using Falconer's (1981) notation, consider a typical genetic locus with two alleles (A, a) under the usual random mating conditions of a population:

Genotype	Frequency	Frequency in inbred lines	Genotype value	Inbred lines frequency x value
AA	p^2	$p^2 + pqF$	a	$ap^2 + apqF$
Aa	$2pq$	$2pq - 2pqF$	d	$2dpq - 2dpqF$
aa	q^2	$q^2 + pqF$	-a	$-aq^2 - apqF$
Population mean: 1.00				$a(p-q) + 2dpq\,(1-F)$

In the above, the reduction of the population mean due to inbreeding (F) is $D_i = 2d_ip_iq_iF$ which is the inbreeding depression for this locus. Summed overall, loci total inbreeding depression is $D = -2F\sum_i d_ip_iq_i$. It can only occur if there are genes with dominance. Secondly, among relevant loci (dfo) there must be a preponderance of unfavorable recessive genes (d positive) so that their effects add up to a negative total. Furthermore, inbreeding effects assume maximum values if relevant loci are at gene frequencies near 0.5, where 2pqd is a maximum.

Of the five species in Table 39.6, the Leghorn chickens show lowest inbreeding depression, Japanese quail and partridges the most. The Leghorn flock in question, Line P in Abplanalp et al. (1964), had been under continuous selection as a closed population for over 30 generations when the above experiments were started. During that time its inbreeding (F) had risen to approximately 30 percent leaving only 70 percent of the original heterozygosity on which renewed inbreeding could act, thus reducing the absolute level of inbreeding in full-sib matings to about (0.7 x .25) or 17.5 percent. However, such prior inbreeding could hardly account for the difference between Japanese quail and chickens.

A second factor in the species differences may be the selection history of the two populations, with Japanese quail perhaps having undergone less intensive artificial selection

Table 39.6. Effects of 25 percent inbreeding on the performance of several gallinaceous species. Inbred performance is expressed as percentage of noninbred performance levels.

Trait	White Leghorn chicken[1]	Broad-breasted Bronze turkey[2]	Japanese quail[3]	Chukar partridge[4]	Ring-necked pheasant[5]
Fertility	99.1	98.8	79.2	61.4	95.0
Hatchability					
Embryo inbred	90.9	83.4	72.6	81.5	84.2
Hen inbred	97.0	92.1	86.3	87.8	--
Viability of hens	94.3	90.7	81.5	99.2	83.0
Egg production	90.4	89.5	88.9	97.6	63.6
Total reproduction	74.4	61.6	35.9	42.5	42.2
Egg weight	100.0	95.9	99.2	99.5	96.6
Age at first egg	100.0	100.0	104.2	98.6	--
Body weight	95.0	89.9	96.4	99.0	--

References: [1] Abplanalp (1974); [2] Abplanalp and Woodard (1967); [3] Sittmann et al. (1966); [4] Woodard et al. (1982); [5] Woodard et al. (1983).

than Leghorns. If this were the case, deleterious genes with relatively large effects would have been reduced in frequency for chickens, with a consequent increase in average noninbred performance and a reduced inbreeding depression according to the formula above. Turkeys, with a relatively recent history of artificial selection for reproductive performance, may fit this last explanation in carrying deleterious genes at still high frequencies.

Inbreeding depression in partridges followed the pattern of Japanese quail closely, even though the flock under investigation had been established directly from a wild population. As such it had no artificial selection history for high egg production nor a substantial level of inbreeding preceding these experiments. However, the numbers of eggs laid by wild partridges are limited to a relatively short period of lay, followed by complete cessation of laying during molting or brooding. These short laying periods are a genetic adaptation to natural reproduction, where the number of eggs in a clutch is limited to allow successful incubation. For such a trait, deleterious genes may be at low frequencies from natural selection, thus resulting in relatively low levels of inbreeding depression.

Inbreeding depression of egg production characteristics in two long-term selected Leghorn populations were recently reported by Ameli et al. (1988). They used full-sib and half-sib matings within a single generation as a means of diagnosing potential problems from inbreeding. Observed relative performance levels of 25 percent inbreds were 99.1 for viability, 92.4 for egg production, 96.7 for hen body weight, and 98.1 for egg weight. All values were thus higher than those shown for the California flock in Table 39.6. The above results support the hypothesis that successful long-term selection for high egg production and reproduction may lead to reduced inbreeding depression of these traits.

Inbreeding Effects Calculated From Intra-line Regressions

In closed pedigreed flocks, inbreeding coefficients can be calculated by the methods of Cruden (1949) if generations are distinct, or by those of Emik and Terrill (1949) if they overlap substantially, as shown in Appendix A. Families of full-sibs then each can be assigned an inbreeding coefficient (F) and the family average ($\bar{y}$) of a production trait regression on F.

Several studies of inbred lines have made use of such regression estimates of family performance averages on inbreeding coefficients. Thus intrasire regressions were calculated by Shoffner (1948) for nine inbred lines of laying hens. Intrasire regressions within lines were also used by Blow and Glazener (1953) and by Wilson (1948a, 1948b) to assess inbreeding depression within lines previously inbred by close matings. These studies closely agree with the previous conclusion that hatchability is the trait most severely affected by inbreeding, followed by egg production. Small inbreeding effects were found for egg weight and body weight of mature hens, in close agreement with results based on full-sib matings shown in Table 39.6. Regression techniques were also used by Tebb (1957) in an outbred Leghorn flock. When compared to deliberate sib matings within generations, the above regression estimates require relatively larger data collections and continued updating of inbreeding coefficients over several generations before reliable estimates of inbreeding effects are obtained.

In a more recent study, Cahaner et al. (1980) investigated inbreeding effects in commercial turkey breeding flocks with seven male lines primarily selected for growth and eight female lines selected for growth and reproductive performance. Their results are shown in Table 39.7 in terms of relative performance for an estimated 25 percent inbreeding. These percentages are extrapolations beyond inbreeding differences observed among families within the flocks in question, since close matings were avoided in female lines and were very rare in male lines. The results in Table 39.7 show that male lines suffered greater inbreeding depression than did female lines in all traits except egg weight. The lower inbreeding effects of female lines suggests that selection for higher egg production, better reproduction, and viability had eliminated deleterious genes from these populations, while male lines selected primarily for growth had retained more of such unfavorable recessives. These results together with the results from commercial Leghorns (Ameli et al., 1988) suggest that inbreeding depression in commercial flocks may be reduced by artificial selection for high reproductive performance.

Estimates of inbreeding effects from intra-line regressions differ from those based on deliberate full-sib or half-sib matings within a single generation in that common ancestors are relatively more remote. Also, in closed flocks differences in inbreeding coefficients may involve relatively few sources of gene identity. Thus a recent pedigree analysis by Nordskog and Cheng (1988) showed a single sire, from a 1965 base population consisting of 55 birds, to be represented in the pedigree of 94.5 percent of 1983 progeny. Of the 55 original ancestors, only two sires and five dams remained as common ancestors, all others having been eliminated by either chance or selection. To what extent selection among descendants of common ancestors affects their true contribution to inbreeding requires further study.

Estimates of inbreeding depression based on intra-flock regressions are subject to large sampling errors because inbreeding coefficients within populations do not vary greatly, especially if close matings are avoided (see Cahaner et al., 1980). Thus, if knowledge about inbreeding effects is important, the use of diagnostic full-sib matings is preferable.

Inbreeding effects observed for Japanese quail under full-sib mating by Sittmann et al. (1966) (Table 39.6) were of a magnitude to make the permanent propagation of inbred lines under continuous full-sib mating very unlikely. Several other experiments have since confirmed this prediction even though selection within and between lines was used to maintain viable lines. Thus Kuhlenkamp et al. (1973) were able to maintain six of 17 initial inbred lines for five consecutive full-sib generations. By strong within-line selection

Table 39.7. Estimated performance levels of commercial male and female turkey lines for inbreeding 25 percent above population level, based on regressions of performance on inbreeding coefficients calculated from pedigrees within lines. (Adapted from Cahaner et al., 1980).

	Performance average of 7 male lines	Relative performance of 25% inbreds, %	Performance average of 8 female lines	Relative performance of 25% inbreds, %
Hatchability				
Offspring inbred	64.8	78.1	68.2	98.0
Hens inbred	64.8	79.7	68.2	86.7
Egg number	71.4	97.4	105.3	98.6
Viability, 17 wks, %	94.1	96.3	97.7	99.5
Body weight, 17 wks, kg				
Males	10.23	96.9	8.27	101.8
Females	7.69	93.1	5.97	101.6
Egg weight, g	84.5	98.9	84.9	95.5

they were able to reduce inbreeding depression in later generations to less than the expected linear decline with increasing calculated inbreeding (F). Similarly Sato et al. (1984a, 1984b, 1984c) were able to maintain three of 16 lines for four full-sib generations. But total fitness, reflecting the survival of adults from eggs set, declined at an accelerating rate with inbreeding. In their study, as well as in those by Kuhlenkamp et al. (1973) and by Sittmann et al. (1966), surviving inbred lines had higher reproductive performance at the outset than lines lost under inbreeding, thus demonstrating the presence of substantial additive between-line variance after the first full-sib mating ($F = 0.25$).

Linearity Of Inbreeding Depression

A separation of selection effects for improved reproductive fitness of lines from the adverse effects of inbreeding is necessary to allow conclusions concerning the linearity of inbreeding depression with F. An experiment which allows a rise in F without attendant selection has been used by Woodard et al. (1983) in ring-necked pheasants, by repeated backcrosses of successive generations of inbred females to the same male. Under this mating plan the selection for survival of females is counteracted each generation by reintroduction of genes from the common ancestor. Thus, inbreeding rises gradually towards 50 percent while the average performance of successive generations approaches that of the common male. The results of two such lines surviving six generations of backcrossing are shown in Table 39.8.

The two lines both reached a relatively constant level of performance after two backcrosses, with the exception of egg number where the decline continued further. However, the results are in good agreement with the hypothesis of a linear decline in hatchability, egg production, and egg size with increases in the coefficient of inbreeding. Since each inbred line derived by repeated backcrossing eventually represents just the common ancestor's genes, the variability between different lines can be used as an indication of genetic variability in tolerance to inbreeding of the population as a whole. Promising ancestors can be discerned as early as the second backcross, according to results of Woodard

Table 39.8. Performance of two lines of ring-necked pheasants, each inbred by repeated backcrossing to a single male for six generations. (Data from Woodard, 1989, personal communication).

			Line C (Chinese pheasant)				
Generation	F[1], %	M[2], %	Hens, no.	Eggs/hen, 12 weeks	Egg wt., g	Fertility, %	Hatch, %
0	0	50.0	7	46	29.9	90.4	53.1
1	25.0	75.0	10	38	28.1	90.6	41.9
2	37.5	87.5	8	23	27.7	84.8	42.3
3	43.8	93.8	6	21	28.9	93.7	40.3
4	46.9	96.9	8	28	26.8	91.1	46.6
5	48.4	98.4	8	22	27.3	83.2	42.3
6	49.2	99.2	2	9	26.5	94.6	42.9

			Line M (Mongolian pheasant)				
Generation	F[1], %	M[2], %	Hens, no.	Eggs/hen	Egg wt., g	Fertility, %	Hatch, %
0	0	50.0	8	42	36.2	99.1	70.0
1	25.0	75.0	9	38	34.9	93.0	52.5
2	37.5	87.5	8	29	33.8	96.1	72.1
3	43.8	93.8	7	27	34.1	95.3	61.3
4	46.9	96.9	9	19	34.0	85.8	52.4
5	48.4	98.4	9	24	34.6	58.2	64.0
6	49.2	99.2	4	32	32.5	78.7	52.0

[1] F = percent inbreeding.
[2] M = percent of genes from common male.

et al. (1983).

In experiments with full-sib mating, the linearity of inbreeding depression can only be tested if selection between and within lines has been minimized, as was the case of results in Table 39.6, and in a study by Cahaner (1984) with broiler chickens. His results for three generations of full-sib mating are given in Table 39.9. Linearity of inbreeding effects can be assessed by examining performance in two steps of 25 percent inbreeding: from 0-25 percent ($\Delta_1 x$) for the first sib generation, and from 25-50 percent ($\Delta_2 x$) from the difference in performance of the first and third generations of sib mating, respectively. Accelerated inbreeding depression is reflected in greater effects for the second inbreeding interval $\Delta_2 x$ and can be measured by the second difference of the two intervals:

$$\Delta^2 x = \Delta_1 x - \Delta_2 x = (x_{50} - x_{25}) - (x_{25} - x_0).$$

Cahaner's (1984) results suggest that all traits examined, with the exception of hatchability, showed an accelerated rate of decline for the interval between 25 and 50 percent inbreeding. Egg size, broiler weight, and age at first egg, which in other studies were found to have mild inbreeding effects, stand out as declining more as inbreeding advances. Hatchability needs to be considered as a special case because maternal inbreeding takes effect only in the second interval. At the same time hatchability probably is under the strongest natural selection pressure of the traits examined. Declining rates of inbreeding depression of hatchability have also been reported by Kuhlenkamp et al. (1973) in Japanese quail, whereas Sittmann et al. (1966) and Sato et al. (1984a, 1984b, 1984c) showed accelerated inbreeding depression for hatchability. Sittmann et al. (1966) also showed accelerated rates of inbreeding depression in viability and age at first egg. Egg production however appears to follow a nearly linear decline in Japanese quail, chickens and ring-necked pheasants.

Genetic models which predict accelerated or slowed declines in inbreeding effects at high levels of F, in the absence of selection, have to be based on the presumption of epistatic gene interactions. A number of two-locus epistatic gene models have been developed by Crow and Kimura (1970). Their most important conclusion was that epistatic interactions between loci with no dominance effects do not deter from linearity of inbreeding effects. Of interest may be a model in which two loci each with deleterious recessive genes show an enhanced inbreeding depression when the two loci are simultaneously homozygous for the recessive gene. In such a situation inbreeding depression depends on both F and F^2. The latter component contributes little at low F values but it dominates the decline when F is high. Another model with potentially enhanced epistatic inbreeding effects is one for loci with multiplicative gene effects.

Because of the pervasive effects of unavoidable natural selection, conclusions about

Table 39.9. Inbreeding depression in production traits of broiler lines at 25 and 50 percent inbreeding with full-sib mating. Performance levels are expressed as percentage of noninbred control matings (= 100 percent). First differences from 0 - 25 percent inbreeding levels and between 50 - 25 percent levels are shown, as well as the second difference $\Delta^2x = \Delta_2x - \Delta_1x$) as an indication of linearity of inbreeding depression. (Adapted from Cahaner, 1984).

	Control noninbred average	Inbred performance % of control				
				First differences, %		Second difference, %
Trait	F=0	F=25%	F=50%	Δ_1x	Δ_2x	Δ_1x - Δ_2x
Hatchability of eggs set, %	79.5	79.7	72.4	-20.3	-7.3	+13.0
Viability, weeks, %	97.1	95.2	84.7	-4.9	-10.5	-5.6
Egg production to 40 weeks	56.2	68.8	35.5	-31.2	-32.3	-1.1
Egg weight, g	57.9	96.4	91.1	-4.6	-5.3	-0.7
Body weight at 7 weeks, kg	1.68	92.4	77.3	-7.6	-15.1	-7.5
Age at first egg, days[2]	191	105.2	121.3	+5.2	+16.1	(+)10.9[1]

[1] (+) Retarded age at first egg.

[2] Data shown under F = 0 are averages of actual measurements = 100 percent.

gene action and interaction contributing to inbreeding depression in poultry remain tentative, especially since no one genetic model enjoys *a priori* or experimental support. However, from the point of view of breeding viable inbred lines, genes which counteract inbreeding depression would be of interest and have been demonstrated. Thus, in the case of the lethal recessive gene causing diplopodia in chickens, Abbott (1959) was able to obtain live homozygous recessives by artificial selection for embryo survival in progeny tests. Similar cases were reported by Landauer (1967), thus proving epistatic effects of additive genes capable of counteracting or correcting deleterious recessive gene effects.

Artificial Selection Against Inbreeding Depression

The role of selection under inbreeding was investigated by Schultz (1953) who analyzed an inbreeding experiment of eight half-sib inbred lines selected for high and low performance in egg number and egg weight. He concluded that selection for egg size produced expected genetic changes, while egg production declined in all lines regardless of selection. Low reproductive rates eventually led to a termination of this study. Lerner (1954) made the observation that birds of the above lines showed an increased incidence of crooked toes. He termed this phenomenon a phenodeviant and postulated that such abnormal patterns of development arose because of a reduced level of homeostatic physiological control. He further postulated that genetic mechanisms such as the superior performance of heterozygotes gave rise to so-called genetic homeostasis, genetic mechanisms for the retention of heterozygosity in populations.

From the point of view of establishing viable inbred lines, it would be of interest to quantify the potential efficacy of artificial selection within partially inbred lines. One such experimental design involves the use of a so-called bottleneck in population size, which will establish an experimental flock with a relatively high but constant level of inbreeding over several generations during which selection can be applied.

An experiment using a bottleneck of three generations of full-sib mating in 16 experimental lines of Leghorns was published by Abplanalp (1988). Inbreeding under this mating plan was thus brought to 50 percent and held at that level for several generations following expansion in flock sizes.

As shown earlier, the expected genetic variance due to genes with strictly additive gene action ($d = 0$) would be reduced within lines to $V_{aw} = V_o(1\text{-}F)$. Selection within lines would thus change performance from additive genes at half the expected rate for noninbred controls. For genes with strictly recessive gene action and low initial frequency, the genetic variance under inbreeding would increase within and between lines, as demonstrated for continued full-sib mating by Robertson (1952). The effect of a bottleneck on genetic variance of subsequent generations closely follows Robertson's (1952) results as shown by Abplanalp (1988).

Figure 39.1 shows expected genetic variances in randomly mated inbred lines for a recessive gene with frequency of $q_o = 0.02$ in the population of origin, following bottlenecks due to full-sib mating from one to nine generations. Maximum increases in genetic variances within the expanded lines are attained by three generations of full-sib mating; hence the use of this design in the above mentioned study.

Artificial selection for increased egg number in the 16 experimental lines after their expansion to $N_e = 10$ resulted in selection responses for inbreds comparable to those obtained with noninbred controls. Six of the inbred lines were selected for 12 generations

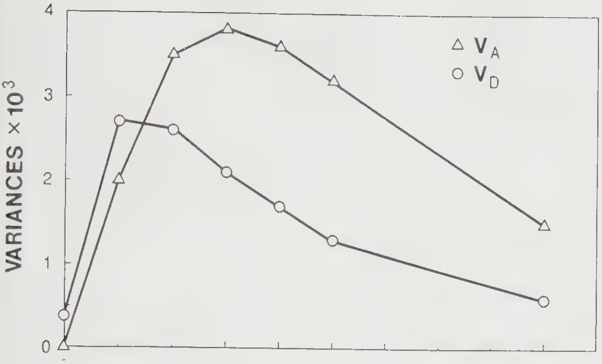


Figure 39.1. Genetic variances due to a recessive gene within lines established by inbreeding from a common base population with full-sib mating from one to nine generations, thus establishing bottlenecks in population size of increasing severity. Inbreeding was then followed by expanding lines to a large size, and variances were calculated on the assumption of random mating in the latter. Numerical values of genotypes: *AA*=*Aa*=0.5; *aa*=-0.5. Gene frequency in base population: $q_o = 0.02$. (From Abplanalp, 1988).

as shown in Figure 39.2, with substantial selection responses. However, the two sets of three lines each differed in their selection history. Lines 54, 56 and 58 (Figure 39.2A) originated from a flock with previous cycles of inbreeding; the other lines (70, 80, 82) derived from a base population maintained under random mating (Figure 39.2B). These latter lines showed greater inbreeding depression at the outset but a faster rate of improvement than lines previously subjected to selection under cyclic inbreeding. Crosses among inbred lines continued to show heterosis for egg production. From these results it can be concluded that selection against recessive genes causing inbreeding depression in egg number was effective. However, deleterious recessive genes fixed at random during the inbreeding phase of the bottleneck remained and gave rise to heterosis of line crosses. Estimates of variance components in the above study indicated that total genetic variability within inbred lines was of equal magnitude to that of inbred control populations.

Another experimental design for selection against deleterious recessives is based on cyclic inbreeding. Under such a plan, one or more generations of close inbreeding, such as full-sib mating, is followed by a cross of unrelated inbreds followed by another cycle of close inbreeding.

With large numbers of sublines within a flock, selection against genes causing inbreeding depression is possible during the inbreeding phase at a relatively constant level of F, as long as crosses among unrelated inbred families are possible. Cyclic inbreeding systems have been investigated for chickens by Abplanalp (1974) and for Japanese quail by MacNeil et al. (1984). In both cases, cycles of intermittent single generation full-sib mating and subline crossing were used. Inbreeding in alternate generations then rises as $F_1 = 1/4$, $F_2 = 5/16$, $F_3 = 21/64$, leading to a limiting value of F = 1/3 or 33 percent. This limit is approached closely in the third cycle, affording an opportunity to estimate selection parameters after that point.

The results of MacNeil et al. (1984) are shown in Figure 39.3 for Japanese quail populations under mass selection and selection with cyclic inbreeding, respectively. In the figure, regression lines are shown for successive index averages of inbred generations (F ~ 33 percent) as distinct from that of cross performance levels in alternate generations (F = 0).

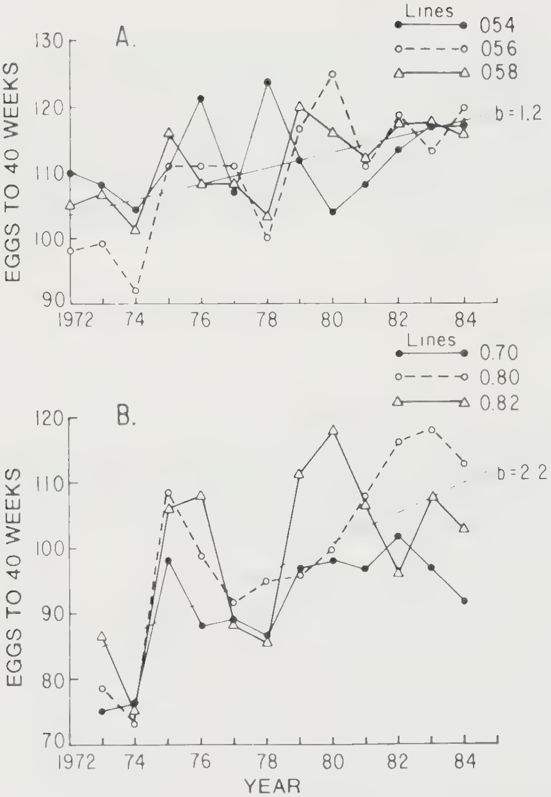


Figure 39.2. A: survivor egg production to 40 weeks of three Leghorn lines (054, 056, 058) inbred by full-sib mating to F=50%, followed by an expansion in numbers to an effective population size of approximately ten. Individual index selection for egg number with constant egg size was practiced after lines were expanded. The three lines were derived from a common base population with index selection for egg number under a history of cyclic inbreeding by full-sib matings.
B: Survivor egg production as in A of three lines derived from a base population with a history of index selection for egg number under random mating. (From Abplanalp, 1986).

The results show that selection progress of inbred performance of cyclic lines equalled or exceeded that obtained with mass-selected controls. Cross performance of cyclic lines also showed progress from selection but to a lesser extent than inbred performance, with a regression of 0.018 vs. 0.056 for inbreds. It should be noted that the regression for inbreds was fitted only after the second inbred generation because inbreeding coefficients of inbreds remained fairly constant from that point on. These results demonstrate that selection against inbreeding depression under sib mating was effective. Since only alternate generations were subject to inbred selection, it could be expected that there would be twice as much response with continuous inbreeding, such as in a line derived from a bottleneck. The results in Figure 39.3 also show that selection under inbreeding improved performance in crosses, presumably due to response from additive genes or from a reduction in the frequency of deleterious genes originally present at high frequencies.

In a computer simulation study of cyclic inbreeding, Madalena and Hill (1972) came to the conclusion that such a cyclic breeding system is effective for the reduction of inbreeding

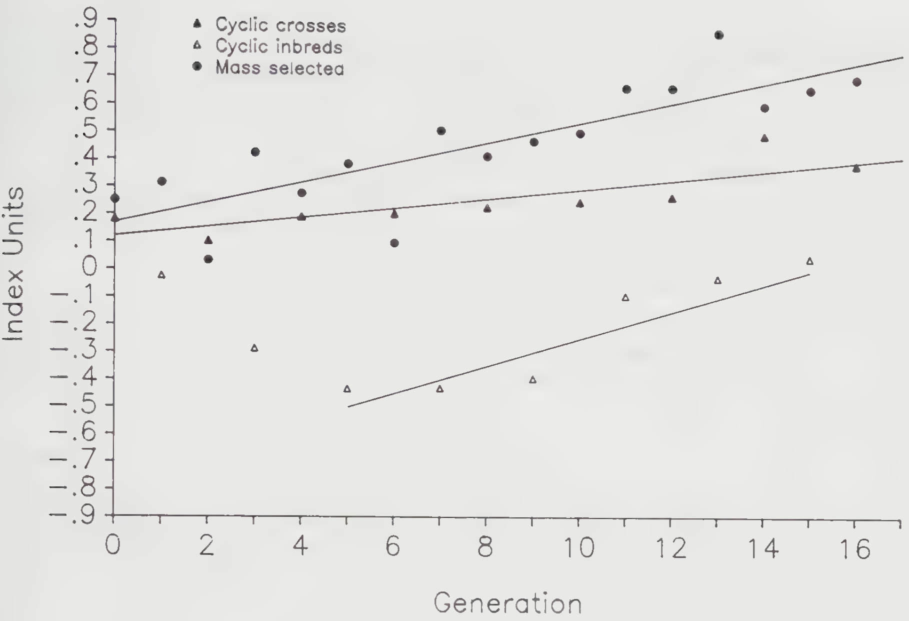


Figure 39.3. Genetic responses of a mass-selected control population of Japanese quail compared to that of a population selected under cyclic inbreeding. In all cases selection was based on an index (I) designed to maximize feed efficiency using egg production (P), egg weight (E), and body weight (B) giving I=(PxE)/B. In the cyclic breeding system, separate regression lines were fitted to inbred generation means obtained from full-sib matings and generation means involving crosses of unrelated inbred sublines. (Adapted from MacNeil et al., 1984).

depression. However, for strictly additive genes inbreeding cycles do not make for improved selection efficiency according to Dickerson and Lindhé (1977).

RETENTION OF HETEROZYGOSITY IN INBRED LINES

Estimates of inbreeding levels (F) may be subjected to experimental test if alleles of segregating genes can be identified at the outset. Blood groups of chickens have been a prominent source of such information. Early findings by Schultz and Briles (1953) showed an excess of heterozygotes over expectations for the A and B blood group systems in inbred lines of Leghorns derived by half-sib matings. They concluded that superior reproduction of heterozygotes was responsible for the maintenance of heterozygosity in these lines.

In a survey of the B-blood group system genes in some 73 lines of chickens, Briles et al. (1957) found only two lines to be homozygous. Sixteen lines with inbreeding over 65 percent were segregating for two *B* alleles each, and of 17 lines inbred 50-63 percent six segregated for three *B* alleles. Of 11 noninbred lines, two had four *B*-types and one as many as eight different *B*-types. Overall a total of 21 different *B*-haplotypes (alleles) could be distinguished serologically. The authors concluded that overdominance effects had caused the retention of multiple alleles in lines with high calculated degrees of inbreeding. A detailed subsequent investigation was made by Briles and Allen (1961) of seven of the above lines, all inbred to levels of 50 percent or higher and segregating for *B*-haplotypes. *B*-

associated differences for egg production within families were found in only three lines, in all cases as a result of one significantly inferior homozygote. Pooled results over lines and two years showed heterozygotes to have equal viability to the better of two homozygotes (53.2 vs. 55.5 percent respectively), whereas the less viable homozygotes had significantly lower viability than either of the other two genotypes (35.8 vs. 47.6 percent). Although the authors favored heterozygote superiority as a possible explanation of their results, the latter when taken at face value suggest complete dominance of the better haplotypes for both traits. However, interactions of genotypes with age and over years may be considered possible causes of long-term superiority of certain *B* heterozygotes.

The B-blood group system has been widely investigated and its effects on disease resistance, viability and hatchability have been documented (see Chapter 22). Dominance of the more favorable *B*-types has often been demonstrated, while heterozygote superiority over homozygotes has been less prominent. Thus, even for this important gene complex overdominance remains only one of several explanations for continued segregation of its genes in closed flocks.

In a similar study Gilmour (1959) found five blood group systems to be segregating in inbred lines reported to have been under continuous full-sib mating for 22 generations. At their calculated inbreeding of nearly 99 percent these lines should have been isogenic with high probability. Gilmour (1959) concluded that the selective advantage of blood group heterozygotes was responsible for his findings, without actually estimating reproductive values of genotypes. A more recent survey of nine inbred lines, after ten generations of strict brother-sister matings (Abplanalp et al., 1981), found observed heterozygosity of nine blood group systems to be in good agreement with expectations based on calculated frequencies of genotype distributions for neutral genes.

The consequences of superior heterozygote reproduction for the retention of heterozygosity within inbred lines have been investigated theoretically by Reeve (1955). Using only within-line selection, he examined the situation where both homozygotes have equal reproductive potential, v(*aa*) = v(*AA*), but where they are inferior to the heterozygote which has a relative reproductive rate of v(*Aa*) = 100 percent. Table 39.10 shows that for full-sib mating the rate of inbreeding approaches 19 percent when homozygotes are fully viable (v = 100) but it is reduced to 1.8 percent when homozygotes have a reproductive rate as low as v = 20 percent. Such low homozygote reproduction at one locus would cause 40 percent of potential offspring to be lost without inbreeding, and would result in substantial inbreeding depression of ten percent for a single generation of full-sib mating. With lesser degrees of heterozygote advantage more genes could be maintained by strong natural selection under inbreeding, but they all would eventually become homozygous in a given surviving line. The results of Table 39.10 also show that for breeding systems with less intensive inbreeding, such as double first cousins (N = 4), the relative reduction of realized inbreeding below that expected for neutral genes is substantially greater than under full-sib mating.

For practical purposes heterosis is unlikely to delay the attainment of homozygosis in full-sib lines (N = 2), but may do so under double first cousin mating (N = 4) or in larger populations. Also, if heterosis were to be the cause of persistent heterozygosity in full-sib lines, its effects should be readily observable in comparisons of homozygous and heterozygous genotypes for a given locus.

The retention of heterozygosis in inbred lines has also been considered for a breeding system where selection between sublines takes place, so that inbred families with poor reproduction are replaced by sublines of more reproductive ones (Hayman and Mather, 1953). Under such a breeding system heterozygosity can be retained indefinitely if v-values

Table 39.10. Increase in inbreeding (%F) for a heterotic locus with equal viabilities of homozygotes (v_1; v_2) expressed as percentage of heterozygote viability (*Aa* = 1.0). Regular mating systems for full-sibs (N = 2), half-sibs (N = 3), and double first cousins (N = 4) are shown after they have reached a constant decline in heterozygosity within lines. (Adapted from Reeve, 1955).

Homozygote viability, % $v_1(AA) = v_2(aa)$	Realized rate of inbreeding, F% Full-sib N=2	Half-sib N=3	Dfc N=4
100	19.1	13.0	8.1
80	14.9	9.3	5.0
60	10.3	5.7	2.4
40	5.7	2.5	0.7
20	1.8	0.5	0.06

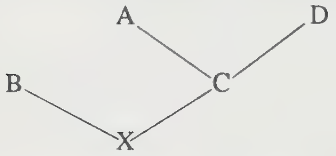
of homozygotes are sufficiently low. Thus for full-sib mating, v-values of 0.76 or less would retain heterozygosity indefinitely, but at a cost of high inbreeding depression and low reproductive performance of noninbreds.

The above considerations make it clear that observations of persistent polymorphisms within inbred lines need to be interpreted with care since the formation of sublines or accidental contamination of lines may be involved, rather than simple heterosis of the loci in question. Also, in the case of blood groups there are important differences in gene function which may make generalizations unattractive.

Although many experiments have been conducted to breed viable inbred lines, the information on gene action derived from such efforts has been modest. Investigations of specific genes with effects on reproductive performance of individuals remain an important future tool for our understanding of inbreeding effects.

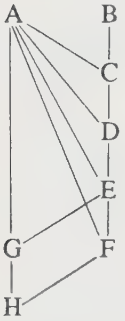
Appendix A. Calculation of Inbreeding Coefficients from Coancestries.

Whenever matings between generations occur, such as with parent and offspring or grandparent with offspring, the coancestry method given by Emik and Terrill (1949) can be used. For a typical pedigree, coancestries can be generated by the formula:



$$F_x = f_{BC} = \frac{f_{BA} + f_{BD}}{2}$$

The pedigree of a mating system to be analyzed is shown below and the coancestry matrix is given in Table 39.11.



Assume A and B to be noninbred and unrelated. All individuals are listed in the order of their appearance over generations. Computations are then started in the upper left hand corner of Table 39.11 and carried out row by row. Since the matrix is symmetrical, only values in the upper right half need be included. For each individual shown at the column head, both parents need to be listed above to permit easy reference to the mating pattern without further use of the pedigree chart. The procedure is as follows:

Step 1: $f_{AA} = (1+F_A)/2 = 1/2$
$f_{BB} = 1/2$

Enter these values in first diagonal for hypothetical self matings.

Step 2: $f_{AB} = f_{BA} = 0$

Enter f_{AB} in second cell of row 1, showing that A and B are unrelated.

Step 3: $f_{AC} = (f_{AA} + f_{AB})/2$
$= (1/2 + o^{A})/2 = 1/4$

Note parents of C are A and B; f_{AA} and f_{AB} are taken from above. Enter f_{AC} in third cell of row 1.

Step 4: $f_{AD} = f_{AA} + f_{AC}$
$= (1/2 + 1/4)/2 = 3/8$

f_{AC} is taken from cell 3 of row 1 and f_{AA} as before (cell 1 row 1). Enter f_{AD} in cell 4 of row 1 and so on.

The table can be filled row by row using coancestries indicated by the individual shown on left and the parents of its mate shown at the column head. In the case of hypothetical selfing (diagonal of table), inbreeding of the selfed individual must be taken into account. For example:

$$f_{DD} = (1 + F_D)/2 = (1 + f_{AC})/2 = (1 + 1/4)/2 = 5/8$$

Table 39.11. Coancestry matrix of pedigree.

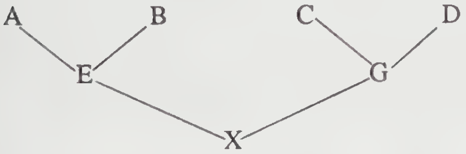
		Parents:		A B	A C	A D	A E	A E
F_A		A	B	C	D	E	F	G
0	A	1/2	0	1/4	3/8	7/16	15/32	15/32
0	B		1/2	1/4	1/8	1/16	1/32	1/32
0	C			1/2	3/8	5/16	9/32	9/32
1/4	D				5/8	1/2	7/16	7/16
3/8	E					11/16	9/16	9/16
15/32	F						23/32	33/64
15/32	G							23/32

From the table the following inbreeding coefficients are obtained:

$F_C = f_{AB} = 0$
$F_D = f_{AC} = 1/4$; $F_E = 3/8$ and so on, taking coancestries of the first row
$F_H = f_{FG} = 33/64$

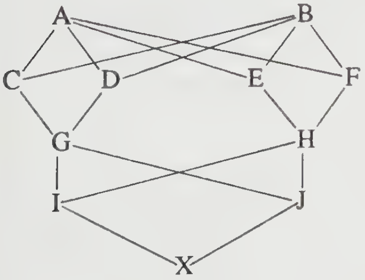
Inbreeding coefficients are also listed to the left of parents (column 1 of the table) to facilitate computation of coancestries for hypothetical selfing.

While the above procedure is perfectly general, another alogarithm based on the average coancestry between both sets of parents of mated individuals can be used. This second procedure is suitable in poultry flocks with distinct generations, as shown by Cruden (1949). Coancestries in successive generations are then computed as follows:



$$F_X = f_{EG} = \frac{(f_{AC} + f_{AD} + f_{BC} + f_{BD})}{4}$$

A worked example is given in Table 39.12 for the case when two sublines derived from full-sib matings are crossed and again inbred. According to Table 39.12:



$$\begin{aligned} f_{CD} &= f_{EF} = (f_{AA} + 2f_{AB} + f_{BB})/4 \\ &= (1/2 + 0 + 1/2)/4 = 1/4 = F_G = F_H \end{aligned}$$

$$\begin{aligned} f_{GH} &= (f_{CE} + f_{CF} + f_{DE} + f_{DF})/4 \\ &= (1/4 + 1/4 + 1/4 + 1/4)/4 = 1/4 = F_I = F_J \end{aligned}$$

$$\begin{aligned} f_{IJ} &= (f_{GG} + 2f_{GH} + f_{HH})/4 \\ &= (5/8 + 4/8 + 5/8)/4 = 7/16 = F_X \end{aligned}$$

Table 39.12. Coancestry matrix using average coancestry between both sets of parents.

Parents			A, B	A, B	A, B	A, B	A, B	A, B	C, D	E, F	G, H	G, H
	Mated birds	(F)	A	B	C	D	E	F	G	H	I	J
–	A	(0)	1/2	0								
–	B	(0)		1/2								
A, B	C	(0)			1/2	1/4	1/4	1/4				
A, B	D	(0)				1/2	1/4	1/4				
A, B	E	(0)					1/2	1/4				
A, B	F	(0)						1/2				
C, D	G	(1/4)							5/8	1/4		
E, F	H	(1/4)								5/8		
G, H	I	(1/4)									5/8	7/16
G, H	J	(1/4)										5/8

Appendix B.

The computation of effective population number from variances in family sizes utilizes four equally important contributions of parents to offspring, namely: sires to sons (M..M) with family mean size n_{mm} and family variance V_{mm}, sires to daughters (n_{mf}; V_{nf}), dams to sons (n_{fm}; V_{fm}), and dams to daughters (n_{ff}; F_{ff}). Furthermore the numbers of male and female offspring retained after selection are usually positively correlated making it necessary to estimate covariances ($Cov_{mm.mf}$ and $Cov_{fm.ff}$). Table 39.13 shows a numerical example for family sizes in a turkey flock with six sires each mated to five dams, and exactly six males and 30 females selected from much larger numbers hatched to reproduce the next generation.

Also given in Table 39.14 are more general formulas applicable when flocks expand or contract in numbers, rather than remaining exactly stable as assumed for the worked case. In Table 39.14 the expected sampling variances in gene frequencies are expressed in units of q(1-q) and computed for each of the four sampling contributions as well as their covariances. It is seen that the estimated effective population size of $N_{ev} = 9.046$ is less than half that expected from random reproduction of the parents $N_e = 20$, due to artificial selection and a consequent increase in variances and covariances of family sizes for selected offspring.

Table 39.13. Family sizes in a flock of turkeys before and after mass selection for large body weight. (Author's data).

Parents		Offspring			
		Before selection		Mated after selection	
Sire	Dam	Males	Females	Males	Females
1	11	3	6	$0 = n_{fm}$	$1 = n_{ff}$
	12	7	7	0	0
	13	2	0	0	0
	14	17	16	0	1
	15	7	4	0	0
		36	33	$0 = n_{mm}$	$2 = n_{mf}$
2	21	0	1	0	0
	22	5	2	1	1
	23	3	1	0	0
	24	0	2	0	0
	25	2	1	0	0
		8	7	1	1
3	31	15	16	2	3
	32	2	4	0	2
	33	5	2	1	2
	34	6	2	0	0
	35	3	2	0	1
		31	16	3	8
4	41	7	4	0	1
	42	4	3	0	2
	43	8	7	0	1
	44	0	2	0	0
	45	11	7	0	1
		30	23	0	5
5	51	11	10	0	3
	52	1	6	0	2
	53	5	11	0	3
	54	1	0	0	0
	55	6	3	0	1
		24	30	0	9
6	61	0	9	0	1
	62	14	12	1	3
	63	3	2	0	1
	64	6	7	0	0
	65	8	15	1	0
		31	45	2	5
Total flock		160	154	6	30

Variances of family size: $V_{mm} = 1.600$; $V_{mf} = 34.000$;
$V_{fm} = 0.400$; $V_{ff} = 1.134$

Table 39.14. Calculation of effective flock size (N_e) for a selected line with N_m sires and N_f dams according to Latter (1959). The worked example is taken from data in Table 39.13. Sampling variances are expressed in units of q(1-q).

Gene passage		Mean family size (n)			Variances of n		Sampling variances Vq [q(1-q)]	
Parent	Offspring	General formula	Stable	Example	General case	Example	General formula	Example
M	M	n_{mm}	N_m/N_m =	1.0	V_{mm} =	1.600	$\frac{1}{16N_m}(1/n_{mm} + V_{mm}/n^2_{mm})$	0.02708
M	F	n_{mf}	N_f/N_m =	5.0	V_{mf} =	34.000	$\frac{1}{16N_m}(1/n_{mf} + V_{mf}/n^2_{mf})$	0.01625
F	M	n_{fm}	N_m/N_f =	0.2	V_{fm} =	0.400	$\frac{1}{16N_f}(1/n_{fm} + V_{fm}/n^2_{fm})$	0.03125
F	F	n_{ff}	N_f/N_f =	1.0	V_{ff} =	1.134	$\frac{1}{16N_f}(1/n_{ff} + V_{ff}/n^2_{ff})$	0.00444
							Covariances of n	
					$Cov_{mm.mf}$ =	5.670	$\frac{1}{16N_m}(2Cov_{mm.mv}/n_{mm}.n_{mf})$	0.02366
					$Cov_{fm.ff}$ =	0.370	$\frac{1}{16N_f}(2Cov_{fm.ff}/n_{fm}.n_{ff})$	0.00789
							Sum of sampling variances = $1/N_{ev}$ = N_{ev} =	0.11054 9.046

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Part V. APPLIED BREEDING AND SELECTION

Chapter 40. INDUSTRIAL BREEDING AND SELECTION

Peter Hunton

The evolution of the poultry industry from a diffuse unorganized system to highly disciplined production complexes took place in less than half a century. It is now a dynamic industry evolving at a rapid pace. This is particularly true of the segments producing poultry meat, due to their continued expansion even after several decades of continued growth.

The development of the commercial breeding industry has accompanied this evolution as a natural outcome. Today's breeding companies could not have succeeded in the context of the traditional system of poultry raising which existed at the beginning of the 20th century. Conversely, the commercial industry continues to benefit from the development of specialized breeding companies.

What have been the major features of this evolution which encouraged the breeding industry to develop? Although the expansion in commercial numbers is important, it is the *intensification* and *concentration* of the industry which has created the environment in which today's breeding system can succeed.

In the early part of the 20th century, almost every small farm possessed some poultry. Many of these small flocks would have self-reproduced and been considered part of the scenery. Productivity was not recorded but was probably extremely low. The birds roamed freely, and provided both meat and eggs in the case of chickens and possibly ducks in some countries. A significant number of poultry keepers in the early days were interested in *exhibition* poultry, breeding for show points, which led to the evolution of many pure breeds. These would have been regarded as specialists, and some of the flocks provided an interesting hobby, if not a reliable income.

The trend to *intensification* and *concentration* was accelerated by discoveries in the fields of *nutrition, housing,* and *disease control.* It was during the first half of the 20th century that most of the vitamins were discovered. Although many of these were identified during research with other species, particularly humans, poultry played a part in these discoveries because of their convenience as experimental subjects. In the 1940s to 1960s enormous strides were made in the understanding of nutritional requirements specifically for poultry. This permitted the formulation of complete diets and the separation of poultry flocks from the free-ranging environment, which had enabled the earlier flocks to obtain many of their nutritional requirements from natural sources. As research progressed, not only the requirements for healthy existence, but the responses to dietary modification over and above minimum requirements became the subject of research and experimentation. Responses in terms of growth, egg production, hatchability, and other traits, enabled poultry producers to become relatively efficient at transforming feed into food. The favorable reproductive rate of many poultry species encouraged rapid expansion to follow these discoveries, and thus, the raising of poultry became a commercial enterprise as well as, or instead of, recreation.

Coincident with the advancing knowledge of poultry nutrition was the development of intensive *housing* systems. While many of these developments resulted from trial and error rather than research, the eventual ability to specify the environmental needs of poultry led to the ability to house birds in previously adverse climates and to produce at all times of the year instead of only in the traditional spring and summer seasons. The empirical discovery that adding artificial light to layers during the winter provided year-round egg production occurred quite early, although the exact mechanisms which support this egg production were not discovered until much later. Nevertheless, the ability to produce eggs year-round gave a tremendous boost to the egg production industry, permitting rational marketing systems, mass production, and the satisfaction of latent consumer demand. In addition to the discoveries in the field of lighting, the development of environmentally controlled housing and the discovery that laying hens performed excellently when housed in cages were major factors in the development of the contemporary egg industry. Similarly, the broiler industry was able to develop when it was discovered that high concentrations of birds could be successfully grown in litter-floor housing. The type of housing varied tremendously, from the partly open housing of the American south to the controlled environment buildings found in northern U.S.A., Canada, and many parts of Europe.

To serve these expanding industries, supplies of *baby chicks* were needed. The farms tended to specialize and were unwilling or unable to continue the system of self-reproduction which was typical for the traditional small flock. While it is possible that each large farm might have developed its own breeding program, there were those who were able to fill this void very successfully. This required not only the ability to develop specialized strains through selective breeding, but also the ability to successfully incubate large numbers of eggs artificially. While artificial incubation has a long history, development of large scale incubation equipment is another feature of the 20th century. Equipment was developed to hatch first hundreds and later hundreds of thousands of chicks of high quality simultaneously.

The development of large scale hatcheries and poultry farms would have been much slower had it not been for simultaneous discoveries in the field of *disease control*. A variety of congenital diseases caused considerable losses during the expansion phase of the hatchery industry. A wide variety of other infectious diseases has afflicted growing and laying birds during the evolution of today's industry. Combinations of disease eradication, chemoprophylaxis, preventive vaccines, and sanitation have been developed which have largely, but not completely, overcome these problems.

Another unrelated aspect of 20th century life has also had a dramatic influence on the development of the poultry breeding industry. The role of *communications* and *transportation* has been extremely important. The *communication* of research results, empirical experimentation, and the publicity afforded to laying tests enabled producers over relatively wide geographical areas to share information, and to apply newly completed research. The publication of laying test data and, later, random sample test information, afforded opportunities for fierce competition among breeders. This undoubtedly sharpened the wits of both breeders and their customers.

The development of reliable *transportation systems* was also responsible for wide-scale dissemination of breeding stock and commercial chickens. Rail systems in North America and Europe placed almost every small farm within the distribution capability of many breeders and hatcheries. The discovery that baby chicks could be shipped over long distances without substantial mortality also contributed to the development of hatchery suppliers to commercial users. As rail networks diminished in mid-century, specialized road vehicles, complete with air-conditioning and ventilation, emerged to replace trains for extended journeys.

The evolution of the commercial jet plane in the period following World War II has also placed world markets at the disposal of those breeders willing and able to service them. It is now entirely feasible to place baby chicks halfway around the world with a high livability record and with delivery within 24 hours of leaving the breeder's hatchery.

FACTORS INVOLVED IN THE EVOLUTION OF THE BREEDING INDUSTRY

Early 20th century breeding methods. In spite of the lack of formalized structure of the industry as it is known today, the poultry fraternity in the early part of the 20th century contained many people who perceived themselves as breeders. Although they lacked most of the characteristics of today's primary breeders, these individuals were involved in selling breeding stock rather than table eggs or meat birds.

At the same time, research workers interested in demonstrating the application of Mendelian genetics to animals as well as plants often chose the domestic fowl as their experimental subject because of its high rate of reproduction. This work has been extensively reviewed by Hutt (1949). While much of the work tended to be academic, some of the findings may have spilled over into breeding practice, particularly in areas such as plumage and skin color.

With the invention of the trapnest, it was possible to distinguish good from poor layers. Even without pedigree information, the unimproved stock available responded to mass selection, based on individual trapnest records (Hutt, 1949). Perhaps of almost equal importance in the development of the early strains was the influence of natural selection. Most of the early breeders refused to use eggs for hatching from pullets in their first laying year. This resulted in natural selection for resistance to disease, longevity, and what the breeders referred to as vigor. Similarly, natural selection for persistency would have taken place since in pedigree pens without trapnests, or even in mass mated flocks, the birds with the greatest persistency would invariably leave the majority of progeny for subsequent generations. In fact, some breeders deliberately selected for longevity, and others attempted to breed only from hens laying at least 1000 eggs. In retrospect, another consequence of these procedures would have been a reduction in the incidence of lymphoid leukosis, due to elimination of mortalities and shedders with reduced egg production.

The main objective of the early breeders was to sell males to other breeders and to those with multiplier flocks supplying hatcheries. While some breeders attempted to maintain closed populations, many would continuously import males from the then fashionable breeders.

Among those who maintained closed populations, there would have been selection based on trapnest records, some modest attempts at progeny testing, and in a few cases, where pedigrees were available, line-breeding or perpetuating individual ancestors either perceived or proved to have superior characteristics. Particularly in the case of males heading pedigree pens, considerable selection emphasis would be placed on breed type and other characteristics observed on careful individual handling.

Although in some ways haphazard, these early breeding activities avoided, to a large extent, the dangers of inbreeding, except for some individual breeders who either accepted the consequences as the price of the system, or who had the skills or good luck to avoid them. As an unpredicted consequence of the frequent exchange of males among breeding flocks, many of the progeny sold commercially resulted from mating unrelated (or only distantly related) parents. One could not call them hybrids but in some cases their performance contained an element of what is now known as heterosis.

On a world basis, the diversity of breeding material was much greater than it is today. There were many breeds in use, and a host of individual strains within breeds. While many of these were grossly inferior in terms of today's commercial characteristics, the system of breeding, and the development of individual breeders led to wide distribution, both of some relatively inferior genetic material, as well as some of the favorable genes which may well be preserved today.

On a local basis, the genetic base was much less diverse. This is because breeders within a region would exchange stock, and the fashionable few would dominate the gene pool for a time because of their popularity and wide distribution.

While a few of the breeders would not have regarded their operations as a hobby, many were little more than that, and comparatively few would have provided for a reliable livelihood. However, in the late 1940s, following World War II, a few of the breeders began to develop poultry breeding as a business, in contrast to a hobby or a sideline. Once the potential was recognized, these breeders began to expand and embark on serious breeding and selection programs. The first geneticist to be employed full time by a breeding company was Dr. W.F. Lamoreux, who began work for *Kimber Farms* in 1943.

Poultry shows. The exhibition of poultry still thrives today, but it is almost totally divorced from the commercial industry. Nevertheless, in the early part of the 20th century, the main way in which breeders could achieve a reputation was by consistent performance at exhibitions. Breed standards existed long before the poultry breeding industry developed. Thus the early breeders would have used these standards as their goals, even before the advent of the trapnest and the emergence of commercial attributes as important selection criteria. Wright's *New Book of Poultry* (1905) specified standards for 98 individual types of 31 separate breeds. Most of the types were distinguished only by plumage color. Many of these breeds survive to this day, but very few of them are involved in the commercial breeding industry. Nevertheless, the development of breed standards and the efforts of the early breeders to achieve conformity with them, laid the foundations of some of today's breeding programs. Such monogenic traits as plumage patterns and colors, skin color, and comb type became fixed in the pure breeds. Whether this is an advantage in the long term is debatable, but it provided uniform material with which commercial breeders were able to commence their genetic selection programs. It is conceivable that greater eventual progress would have been made had the original stocks not been so uniform. However, the impact which this has had up to the late 1980s is probably very small.

Standard laying tests. As the trend towards a commercial poultry industry structure gathered momentum, it became obvious that some form of testing procedure would be necessary in order to identify superior stocks. This led to the development of the standard laying test. In these tests, birds were submitted to the testing station at point of lay, or sometimes after they had begun to lay and been subjected to a short period of trapnesting. At the standard laying test, each pen of birds was separately recorded for approximately 12 months. Although attempts were made to maintain uniform conditions, there was no replication, and major environmental effects influenced the performance of individual pens. Sample size was small, usually no more than 13 birds. The ability of the breeder to grow and provide at point of lay a successful pen was another nongenetic factor which contributed to the result. Opinions vary enormously as to the efficacy of the standard laying test. Some people believe that because of the failure to allow for pullet rearing environment and the variation between pens at the testing station, the results gave little indication of the genetic merit of the stocks involved. Others hold that since many of the environmental effects would be random from year to year, consistent performance in these tests would be a reliable index of the merit of each breeder's product. Of one thing there is no doubt: many reputations were greatly enhanced by consistent performance in standard laying tests over many years. Another criticism of the standard laying test was that the breeder could enter superior birds in the test and use the results to enhance the sale of unrelated or inferior stocks. While there is little doubt that this occurred, the breeders who consistently did well in the standard test would still have been a better choice in terms of stock selection, compared with those who did poorly, or who only occasionally rose to the top.

Random sample tests for layers. While the standard laying tests continued to operate for almost half a century, beginning in the 1950s they quickly gave way to the random sample tests which were specifically designed to test commercial egg laying stocks from the breeders who were beginning to distribute them at that time. According to Marble (1961), the first standard laying test was held in England in 1897, and the first random sample test in the Netherlands 30 years later. The most important single difference between the standard test and the random sample test lies in the method of sampling the entry. In the standard test, mature birds were selected by the owner on whatever basis he felt gave the entry the best chance. For random sample tests, the entry is selected by a disinterested party, either as hatching eggs or newly hatched chicks. Elaborate precautions are taken to ensure that the random sample is indeed representative of the population which it is intended to assess. Additionally, the sample size for the standard laying test was usually 10-13 birds, while for random sample tests the number of pullets is usually at least 50 and frequently several hundred. Because random sample tests usually operate as a public service to breeders and to the industry, they go to considerable lengths to ensure that management, housing, feeding, etc. at the testing station are representative of and transferrable to the commercial industry.

While for the most part these aims are accomplished, there are some unavoidable difficulties which should be described. No provision can be made, or account taken, of the effects of maternal immunity to certain diseases. When progeny originating from parents with different maternal immunity status are mixed, unexpected and, from the viewpoint of comparing genotypes, irrelevant differences in performance may be observed. Infection with infectious bursal disease serves as an example. Progeny from nonimmune parents would be at risk, while those from immune parents may survive a field challenge. In commercial practice, mixing of stocks from different origins tends to be avoided, and account is usually taken of maternal immunity status.

Another example of random sample tests failing to mirror commercial conditions is *M. gallisepticum* status. Most chicks are supplied *M.g.* negative, and management of test stations can generally maintain this status. However, many commercial egg farms around the world are *M.g.* positive. If genotype x environment interactions between production traits and tolerance to *M.g.* infection are important, this may erode the credibility of random sample test results.

After early beginnings in Europe, random sample tests became widespread around the world in the 1950s and 1960s. In 1963-64, 21 separate tests were listed in the United States Department of Agriculture *Report of Egg Production Tests in the United States and*

Canada. However, from about this time on, the numbers of such tests in North America diminished as funding problems and a smaller number of potential entrants took their toll.

From 1963 until 1978 the Agricultural Research Service of U.S.D.A. published two-year combined summaries of random sample tests. These summaries were used by many producers as an important consideration in determining choice of strains. Both the summaries, and perhaps more importantly, the individual test results, were also widely used by the breeders in their advertising and promotional efforts. The combined summary provided a much sounder basis for decision making from a statistical viewpoint than any individual test result. Although few producers would have understood the statistical methods used, all could understand the final tabulated data with the confidence limits included. It is probably true to say that the mean values received more attention than the confidence limits. However, this document became an important part of the egg production industry for about a decade until the final publication in 1978. The eventual demise of the publication resulted from the much reduced number of tests available; in 1987 only one test remained in the North American continent. Several random sample tests still existed in Europe and other parts of the world, but their importance and influence were much less than during the period 1950-1980 when they were regarded as a significant factor in the egg industry. The status of European tests was reviewed by Hartmann (1985). One aspect of the development of the breeding industry for egg laying stocks can be well demonstrated by examining the list of entrants in the early years of these U.S.D.A. reports, and again in the last one. The number of apparently independent breeders represented in the 1966 report was 76; in 1978 this number was reduced to 12; a decade later several more of these breeders had disappeared.

Random sample tests for broilers. Public testing of broiler stocks never achieved the status of the egg laying tests. This was partly due to the fact that true random sampling of broiler chicks is difficult to achieve; it is comparatively easy for the entrant to submit large numbers of chicks from specially selected parent stock or hatching eggs. Some tests attempted to circumvent this by random sampling the parents, and then testing successive batches of chicks. This process took a total of 18-24 months, by which time many parent stock buyers would have made another choice. Meanwhile significant genetic changes may also have been made by the breeder.

DISEASE CONTROL

Disease control has always been an important component of the poultry breeding industry for several reasons. Even in the early days of poultry breeding when many thousands of hatcheries were operating in the industry, the dissemination of disease with baby chicks was recognized as a serious hazard.

Pullorum disease. Of the conditions known to be transmitted in baby chicks, certainly the most dramatic is pullorum disease, caused by the bacterium *Salmonella pullorum.* It is egg transmitted. Thus, infected breeder flocks could be responsible for widespread transport of the disease, and consequent high mortality among baby chicks. Before control measures were devised, the carrier state among parent stock was quite common, and the parents themselves did not appear to be diseased. The development of the rapid plate agglutination test to identify these carriers led in many countries to the development of public policy to reduce and eventually to eradicate pullorum disease. Testing programs to detect and eliminate carriers were the foundations for the National Poultry Improvement Program (N.P.I.P.) in the United States, the Poultry Stock Improvement Program (P.S.I.P.) in Great Britain, and many other national programs. As a result of these efforts, pullorum disease has been largely eradicated in many countries, although it still occurs in parts of the world where relatively unorganized poultry flocks exist and reproduce at random.

Disease prevention. Another aspect of the breeder's responsibility in the control of poultry diseases became evident with the widespread distribution of parent stock in the decades following World War II. The fact that poultry breeding stock could be and was shipped around the world from a relatively small number of distribution points meant that any unrecognized or latent diseases could be similarly distributed. While congenital diseases passed from parent to offspring were clearly the most important target for such concern, the causative agents of other diseases might equally well be conveyed along with parent stock to remote destinations. Thus, the commercial breeding companies became acutely aware of the importance of disease control among their primary breeding stocks. This led to the use of elaborate sanitation and isolation procedures at primary breeders' premises. It also resulted in duplication of important breeding stocks at several geographically separate locations.

Yet another aspect of disease control must also be considered by breeders. It is well recognized that susceptibility or resistance to many, if not all, poultry diseases is under some degree of genetic control. Hutt (1958) has written in detail on this subject, and considerable research published since then has served only to confirm his eloquently stated hypothesis. Breeders have applied these results, with more or less enthusiasm, depending on individual circumstances.

National programs to improve the quality of baby chicks distributed by hatcheries were developed in many regions of the world. These programs were particularly aimed at the congenitally transmitted pathogens, initially *S. pullorum*, and later some of the *Mycoplasma* species, especially *M. gallisepticum.* These programs consisted of the registration of hatchery premises, and the systematic testing of all parent flocks. The rapid plate agglutination test for *S. pullorum* became the almost universal tool for the control of pullorum disease. Various tests were used to identify *M. gallisepticum* and other *Mycoplasmae*. Eradication of these organisms from breeding flocks was thereby made possible. Some of these programs, for example the P.S.I.P. in Great Britain, also included methods for systematic upgrading of the genetic potential of the stock being distributed, but these were quite quickly overtaken by the efforts of individual commercial breeders. However, the disease control components of these programs were of considerable and long lasting importance. In addition, the testing and certification procedures in national programs became valuable for breeders wishing to export parent stock, when they came to meet the standards of health control established by importing countries. Most hatcheries in the developed world now distribute stock free of *S. pullorum*, *M. gallisepticum*, *M. synoviae*, and *M. meleagridis* in the case of turkeys.

EMERGENCE OF BREEDING COMPANIES

The combination of technical capability, demand for health control, and genetic improvement encouraged the emergence of dedicated breeding companies, employing their own specialists in genetics, disease control, and other disciplines. This situation was well described by Carter (1964). The specialist breeders distributed parent stock and sometimes grandparent stock through various channels, for multiplication to produce commercial chicks for broiler and egg production.

However, in the 1970s, some of the broiler production companies expanded to such a size that they were able to justify in-house breeding programs to produce their own parent stock. This was accomplished either by establishing entirely new programs, or by acquisition of existing breeding companies. Such integration of primary breeding and selection with commercial production is especially appropriate in the broiler industry. The nature of the selection process is such that large numbers of broiler age birds are rejected, and the existence of a ready market for them is important in determining the overall cost of the breeding program.

Thus, in some respects the industry has come full circle, starting with the separation of breeding from production for reasons of specialization. Companies in both segments expanded, and eventually production companies became so large, and their demands so precise, as to require and sustain their own breeding programs.

BREEDING FOR EGG LAYING STOCKS

Very early in the development of scientific poultry breeding, companies began to specialize in either egg- or meat-type birds. Subsequently, by means of expansion or by corporate mergers, many corporations became involved in all sectors, but the genotypes used and the breeding strategies followed are quite distinct.

Stocks intended for commercial egg production are selected according to a very specialized set of criteria. Considerable research has been conducted into the general area of breeding for egg production. This has been reviewed by Arthur (1986), who also interpreted the major findings in terms of commercial breeding practice. It is worth reviewing one of the main differences between research and practical selection, since this has a major influence upon the design and success of commercial breeding programs. Pure research is planned and designed to test a hypothesis or to determine the results of a particular and precisely specified set of actions. On the other hand, breeding companies must identify commercial goals and achieve them in the shortest possible time. Meanwhile, some of these goals may change in the course of the development program. While a researcher may have a preferred outcome for an experimental program, any result constitutes knowledge and may be published. For the breeder, only one outcome is acceptable: an improved and saleable product.

Selection Criteria

The identification and setting of goals is in itself highly complex. Most breeding companies are involved in developing stocks to serve many markets in different parts of the world. They attempt to do this either with a single product or with a very limited range of products. Each separate breeding program adds significantly to total cost and too much proliferation reduces the eventual progress possible from any one segment.

An example of a trait for which a setting of goals is complicated is egg weight. In some developing countries, eggs are not graded by size but are sold on a per egg basis. In other countries, eggs are sold by the kilogram regardless of their weight. In most North American and West European countries, eggs are classified by weight into a number of categories, seven in the E.E.C., and in North America four to six depending on the exact locality. Price differentials vary between egg weight categories. In much of North America, eggs exceeding 56 or 57 g attract no additional payment most of the time. Conversely, in parts of Europe, a premium usually is paid for eggs weighing over 70 g. This creates a dilemma for breeders. Should they aim to increase potential egg weight until they achieve levels meeting the highest demand anywhere, or aim at an intermediate level, and attempt to achieve maximum levels by means of environmental manipulation?

Another problem in goal setting might be called 'the constantly moving target'. In a highly competitive industry each breeder must avoid placing himself at a perceived disadvantage to others. Egg shell quality is an example of a trait which has fluctuated in importance among breeders at different times. As the commercial egg industry developed and was systematically transformed from many small labor-intensive farms to comparatively few very large ones, mechanization became widespread. Eggs collected mechanically can be subjected to many stresses or insults which would never have occurred when they were gathered by hand. These changes caused major shifts in breeders' priorities. Egg shells which withstood the insults imposed by hand gathering failed when confronted with mechanical systems. Small and previously unimportant differences between strains and between individuals became much more important in this new environment. The goals of some breeding programs had to be rapidly shifted to meet these new demands.

Egg production. Egg production has always been the cornerstone of the selection criteria applied to egg laying stocks. A variety of measures is known to contribute to lifetime egg production. In most cases, although it is recognized that stocks may be molted and lay for more than one season, breeders have concentrated on egg production to approximately 500 days of age. This implies a single year's production, following approximately five months of rearing. While rate of lay is probably the most important contributing factor to this trait, both mortality and sexual maturity are also important components. Because all hens placed in the laying house contribute to the cost of the flock, many commercial operations base their judgements on hen-housed egg production data. This is the total number of eggs produced, divided by the number of hens placed. Thus, substantial mortality will detract from this statistic, even though surviving hens may lay at an acceptable rate. Because the genetic factors influencing mortality are in many cases different from those influencing egg production, breeders have tended to separate these two traits and apply different selection procedures. Most breeding programs are undertaken in favorable near-optimal environments; therefore mortality tends to be low. Selection against mortality under these circumstances is difficult. While families with high mortality may be identified and rejected, this may be inadequate to provide a high degree of general disease resistance. Selection for disease resistance with deliberate exposure has been used to improve resistance to specific diseases, e.g. Marek's disease (see below). General disease resistance has more often been approached by testing potential commercial stocks under field conditions.

Selection for egg production is usually based either on the number of eggs to a fixed date, or on some calculated parameter such as hen-day percent production.

The decision on exactly which selection criterion to use is extremely important. The long-term studies of Gowe and his colleagues at Agriculture Canada in Ottawa have shown that where the selection criterion is egg number per hen housed to a fixed age, the trait most affected is age at first egg rather than rate of production *per se* (Gowe and Fairfull, 1985). On the other hand, where selection is based on rate of production following first egg, it responds directly to selection. Of course, both early maturity and high rate of production are desirable goals. It may, however, be preferable to include both as separate components of an index, rather than to rely solely on selection for egg production to a fixed age to provide the required progress.

The influence of sexual maturity on egg production is obvious; earlier maturing birds, if held to a fixed age, have more opportunities to lay than those which mature later. This is recognized in the derivation of selection criteria by considering age at first egg as a separate trait, or by properly weighting the egg production in the early periods in a total selection program.

Because of the favorable effect on the generation interval, most breeders base a large part of their selection program on part records of egg production. This procedure is based on the assertion of Lerner (1958) that the reduction in generation interval made possible by selection of parents at approximately 40 weeks of age outweighs the added precision of a full year's record in attaining the goal of improved egg production over the first laying year. Proponents of this system claim that even if egg production in the last part of the laying cycle remains constant, improvements to the early part will provide total progress superior to other methods involving longer recording periods. However, since many stocks have very high levels of production for the part record, selection systems are being devised to take into account persistency, with a view to increasing egg production in the second part of the cycle as well as the first. In many cases, a two-stage selection procedure is followed in which retrospective rejection of birds based on their part record may take place.

A detailed study of genetic parameters for egg production, broken into six time periods, was reported by Flock (1977). These data were derived from commercial populations after more than 20 generations of reciprocal recurrent selection. The conclusion was that the optimum length of the egg record period in these populations, for maximum gain in annual production, lay somewhere near 24 weeks. This permits the use of a 52-week generation interval which has other practical attractions. Where production persistency or egg shell quality late in the laying period receive more emphasis, the optimum record length may be increased to between 36 and 40 weeks.

In general, such optima vary with a number of other considerations including at least the following:

- Selection history of the population(s).
- Specific genetic parameters for each population.
- Other traits under selection.
- Selection intensity.
- Reproductive capability of selected individuals.

Egg weight. Egg weight has been identified as a difficult target because of varying demand in different markets. Breeders usually measure the weight of a sample of eggs from all individual birds in the breeding program when they are between 30 and 40 weeks of age. Mature egg weight, from sampling at 60-70 weeks, may also be assessed. The selection emphasis placed on this characteristic varies widely according to the market being served and the geographical area in which commercial birds are being distributed. Most breeders attempt to hold egg weight at current levels and would apply slight upward selection emphasis. This is done to offset the tendency of natural selection to reduce egg weight (Flock, 1988, personal communication) in the absence of artificial selection.

Because egg weight changes with age, some attention has been given to the shape of the relationship between egg weight and age. This is because in almost all markets eggs in the lower weight categories are less in demand than those of greater weights and the birds tend to lay the low weight eggs when their rate of production is at its highest. Thus it would be advantageous if the age x egg weight curve could be altered so that a rapid increase is followed by an almost flat profile once the desired egg weight has been achieved. Although small variations between strains exist, empirical studies have indicated that prospects for making such a change by within-line selection are not favorable.

Body weight. The body weight of egg layers is important for two reasons. It is quite closely related to egg weight with heavier chickens producing heavier eggs. It is also related to overall feed efficiency, since the heavier hen has a higher maintenance feed requirement. Body weight of layers is usually assessed prior to production, and once or twice during the laying year. Over time, most laying strains have been reduced in weight genetically, while attempting to maintain or increase egg weight characteristics.

The traits outlined above will all contribute directly to overall profitability. Many breeders combine these traits in various ways, either in the form of a formal selection index, or to produce some estimate of income over feed cost using arbitrary estimated costs and value.

Egg quality traits. While characteristics related to egg quality are not always incorporated in selection indexes, they are nevertheless important components of selection programs to improve laying hens. *Egg shell quality* was mentioned earlier as an example of a trait which received added emphasis as a result of changing commercial practices. Although some aspect of egg shell quality is usually the selection criterion, the minimization of cracked and broken eggs in commercial systems is the eventual target. Some breeders approach this by actually candling samples of eggs from individual hens to identify cracked shells. However, since the incidence of cracks under these circumstances is often low, most breeders have to resort to measurements on the actual egg shell in an attempt to predict those which might break in commercial circumstances. A variety of techniques to assess egg shell quality was reviewed by Hunton (1985) and some of these have been adapted by individual breeding companies. Direct measurements of *shell thickness* on eggs sampled from the flock are routinely used by some breeders. The estimation of the *specific gravity* of the whole egg, as an index of shell thickness, is perhaps the most commonly used criterion among commercial breeders. The specific gravity may be estimated by the flotation method in which eggs are immersed in saline solutions of ascending specific gravity. The egg is assigned a specific gravity value equal to the solution in which it floats. Alternatively, the classical method of weighing the egg in air and then in water may be used. Other methods include the estimation of *breaking strength*, *nondestructive deformation* using the method of Schoorl and Boersma (1962), and various *puncture tests*. While many of these measurements do not show really strong correlations with field breakage, they have the advantage that measurements are possible on individual eggs. This makes them suitable for inclusion in selection programs.

Egg shell color is an important characteristic and completely separate breeding programs exist for the production of white and brown shell eggs. These are based on different original breeds; the white egg layer is based almost exclusively on various strains of White Leghorn, while brown egg varieties are related to a much more varied pure-breed origin. These include Rhode Island Red, Barred Plymouth Rock, Australorp, New Hampshire, and synthetic strains derived from these and other breeds.

Within the White Leghorn, minor variations exist in the purity of the white shell and some breeders take measurements of this characteristic, usually by the method of percentage reflectance described by Hunton (1962) and Gowe et al. (1965). This method is also used to evaluate brown eggs.

There is considerably more variation in egg shell color within and between the brown egg strains. In most breeding programs measurements are taken of individual eggs during the process of evaluation for other egg quality traits. In some markets, demand for and price is greater for more intense and uniform brown pigmentation, and this becomes included as a major selection goal.

Egg internal quality is another trait which assumes different importance in different markets. In most cases, the criterion used is the Haugh unit, although some breeders use other indices incorporating *albumen height*. For most markets, some minimum standards exist which eggs must achieve. In most cases these would not suggest major emphasis on albumen quality in breeders' selection programs. Nevertheless, breeders must maintain a monitoring program to ensure interior quality does not deteriorate over time.

When eggs are broken out for examination of albumen quality, they are also evaluated for the presence of inclusions, mainly *blood* and *meat spots*. These inclusions usually exist at low frequency; however, meat spots are a particular characteristic of brown shell eggs and many breeders include these as selection information.

Feed efficiency. Feed efficiency in egg layers is of extreme importance. For many years it was assumed that adequate progress in feed efficiency would be accomplished by gradually lowering body weight, and thereby reducing maintenance requirement. However, research by Fairfull and Chambers (1984) showed that even when egg mass output and body weight maintenance and changes were taken into account, residual variation existed in feed efficiency. Thus several breeders have developed facilities for measuring the feed intake of individual hens during the course of egg production, and include this as a separate selection criterion in the index or total profit evaluation.

Disease resistance. Disease resistance presents breeders with a complex selection characteristic. Most breeding company facilities are maintained as isolated minimal disease environments, and tend to experience comparatively low levels of mortality. Thus, it is conceivable that birds might be developed which would be highly susceptible to diseases encountered in commercial situations. While there is good evidence that this is not a major problem, breeders nevertheless pay particular attention to specific diseases where genetic resistance is known to be important. An example is *Marek's disease*. Cole (1968), using populations derived from commercial random sample test entries, showed that rapid genetic changes resulted from selection under deliberate exposure. After three generations, a line selected for resistance had a mortality level below ten percent, while a line selected for susceptibility showed over 90 percent loss when exposed to the same challenge. At the time this work was reported, no commercial Marek's disease vaccine existed. Thus several breeders began to apply this work by deliberately exposing subpopulations of their main breeding lines to Marek's disease infection at specially developed facilities. Selection of the parents or sibs of these exposed birds was then used to increase resistance in the main populations. Practical results of such a strategy in commercial populations were reported by Flock et al. (1975).

Subsequently, the work of Briles et al. (1977) showed that in many cases this genetic resistance was related to a specific haplotype at the major histocompatibility complex locus. Since identification of this haplotype represented a much more economical selection method, some breeders have applied this to commercial populations. The specific haplotype does not occur in all populations. In its absence, other genetic factors still have a major influence on Marek's disease resistance. It has a significant commercial advantage, since Gavora and Spencer (1978, 1983) have shown that even when vaccines are used genetically resistant birds are superior to susceptible ones in the face of high-level challenges with field strains of Marek's disease.

Resistance to *lymphoid leukosis* has also been shown to have a genetic basis (Crittenden, 1968). In fact, single genes seem to exist for resistance to cellular infection by several of the subgroups of the lymphoid leukosis virus. Breeders examined many pure lines for these alleles and where the resistant ones were found, selection has been possible. However, many lines of White Leghorn were found to be homozygous for the susceptible alleles, and therefore different approaches to the control of lymphoid leukosis had to be found. The development of the ELISA test made it possible to identify birds shedding the lymphoid leukosis virus by examining vaginal smears or egg albumen. Thus, rather than selecting for genetic resistance, birds are examined and shedders rejected in an attempt to eradicate this virus from populations. Since vertical spread is the main method of transmission, and horizontal spread while possible occurs only slowly, this system should provide for major reductions in the incidence of lymphoid leukosis.

Single gene traits. Several breeders of White Leghorn stocks, and virtually all brown egg breeders, have incorporated sex-linked genes into their crosses to permit differentiation of the sexes immediately after hatching. In the brown egg stocks, *color sexing* is practiced, the genes most frequently used being the dominant silver (S) and recessive gold (s^+) genes. In the appropriate genetic background, chiefly requiring the columbian pattern, mating a gold male (s^+/s^+) to a silver female ($S/-$) will yield gold pullets ($s^+/-$) and silver cockerels (S/s^+) which can be easily distinguished in the hatchery. Alternatively, sex-linked barring (B) and nonbarring (b^+) may be used.

In the White Leghorn, the sex-linked slow and rapid feathering genes (K, k^+) have been utilized to permit *feather sexing* in the hatchery. Homozygous fast feathering (k^+/k^+) males are mated to slow ($K/-$) females, to yield fast feathering ($k^+/-$) pullets and slow (K/k^+) cockerels in the progeny. Some field experience, and the report of Harris et al. (1984), linking the use of the k^+ gene with apparent increased incidence of lymphoid leukosis, has limited the application of this system of sex differentiation. The exact mechanism whereby the k^+ gene interacts with the avian leukosis virus and the immune system is not clear (Bacon et al., 1986). It may be connected with the presence of an endogenous virus (*ev21*) associated with the k^+ gene, described by Bacon et al. (1985).

While the sex-linked dwarfing gene (*dw*) has been incorporated into some commercial layer lines, none of these have ever reached the market. While under some economic circumstances the dwarf White Leghorn may have been commercially feasible, its delayed maturity, reduced egg production and lower egg weight have served to maintain it as an experimental curiosity.

Selection Methods For Egg Laying Stocks

The selection and breeding methods used by producers of egg laying stocks have varied during the time that they have been in business. Initially, some attempted to mimic the plant breeders of the day, developing highly inbred lines and then identifying superior crosses for sale. Others, from early beginnings, identified superior crosses and then applied selection for additive genes to the base lines to achieve further improvements. A few breeders adopted a system called reciprocal recurrent selection, designed to enhance both additive and nonadditive genes in improving the end product. These methods, and some of their implications in relation to commercial breeding, were reviewed by Arthur (1986) and

Hartmann (1988).

The system of inbreeding and crossing has been largely abandoned. Cost of developing large numbers of inbred lines is comparatively higher in poultry than in the plant species where the system was originally so successful. Furthermore, once superior crosses have been identified there is usually little scope for further improving the end product except substituting lines as improved crosses are identified. This made management of these birds in the commercial industry more difficult, and subject to frequent changes as the genotypes changed.

Even where the concentration of additive variation is the most hopeful pathway to future improvement, it is accomplished in different ways.

Because many of the commercial breeding companies began as relatively small independent breeders, selection systems oriented around closed populations or 'pure lines' tend to predominate. This is the case even though the commercial product is produced by crossing several of these lines in a specific way. Selection within lines has the virtue of simplicity and evolves easily from the mass selection programs which may have preceded the more sophisticated full-pedigree multi-trait operations.

Three- and four-way crosses. Most commercial birds today result from the crossing of three or four pure lines, as illustrated in Figure 40.1.

In the three-way cross, for example, there are two alternatives for the improvement of male line A. Some breeders concentrate on straightforward additive selection within line A. Alternatively, one can attempt to improve line A based on crossbred progeny of the type to be marketed as commercials. Pure-line selection, or a combination of the two methods, is usually used to improve the female lines B and C in the case of three-way crosses or C and D in the case of four-way crosses.

Maintenance of pure lines. Virtually all pure lines used for egg production breeding are maintained as fully pedigreed populations. The primary reason for this, of course, is that most of the traits are expressed only in the females; males must therefore be selected based on the performance of their relatives — either progeny, full-sibs or half-sibs. This adds greatly to the cost and complexity of the breeding and selection operations, but allows much more effective selection than any system of part or nonpedigree identification. It also permits matings to be made so as to minimize inbreeding, an important consideration in most breeding programs which seek to limit loss of variation through inbreeding in pure lines.

Population sizes used by commercial breeders are usually large, with each generation involving several hundred females and 50-100 or more males used for pedigreed

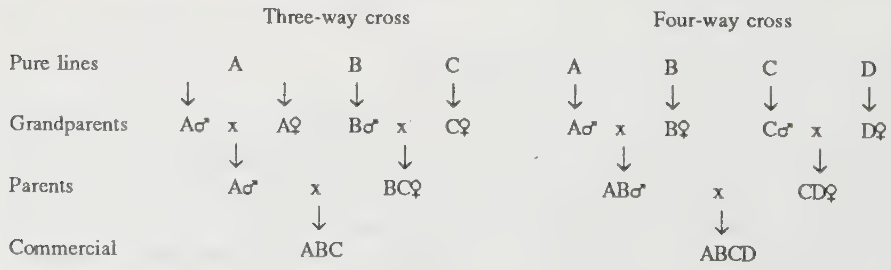

Figure 40.1. Schema for three-way and four-way crosses to produce commercial poultry.

reproduction. In addition, for reasons of security and continuity of production, duplicate lines are frequently maintained at different locations, and even in different countries. This increases the effective size of the population, and also provides opportunities for periodic recombining, or testing of different subpopulations to determine whether divergence has occurred between them. The experience is frequently that such divergence occurs, and this may be harnessed by recombining the lines as a two-way cross, thus reducing the inbreeding in the resultant synthesized new line.

Reciprocal recurrent selection. The system known as reciprocal recurrent selection, originally described by Comstock et al. (1949), became almost a trademark of one particular breeding company. The experience of this company was described by Heisdorf (1969). This system attempts to capture both additive and nonadditive improvements in the end product, by selecting the pure lines based on the performance of the appropriate reciprocal crosses. The system depends for its success on the wise choice of the original lines, as well as on selection progress generation by generation. Theoretical evaluations of this system have shown that overall genetic progress is slower than pure line selection in the early years, but accelerates later. Thus, if the nonadditive improvements in the early period are not as great as expected, the system might be vulnerable to competition from more rapidly improving products resulting from strictly additive selection programs. A review of the theoretical considerations, and some experimental work, is given by Bell (1972).

Use of cages. Nearly all measurements on egg production stocks are taken in individual cages. A few locations exist where birds are tested on the floor, using trapnests, but since virtually all commercial hens are now managed in cages, the measurement of traits in primary breeding stocks in a similar environment is a necessary procedure. In addition, the use of individual cages makes many of the recording tasks much easier and reduces the chances of birds and/or eggs being incorrectly identified. With the advent of computers for assembling and manipulating data, individual cage recording has taken on a new meaning with the use of bar codes, computerized reading devices, direct entry onto disk files, etc. While these technologies have eased the burden of data acquisition and processing, they do not come without cost, and this part of the egg layer breeding operation is one of the most expensive in the animal breeding business.

The use of cages has also necessitated the widespread application of artificial insemination in the pure lines used for egg production stocks. This permits greater flexibility in mating ratios than would be the case for natural matings. There is always the risk that long-term breeding by A.I. in cages might reduce the ability of birds to mate naturally under floor management, which still exists for most commercial breeder flocks. There is no evidence that this has taken place, but it remains a possibility. A few attempts have been made to manage breeding flocks in cages, both with A.I. and natural mating. While A.I. can be and is used commercially, natural mating of laying birds in cages has not always provided maximum fertility and success appears to vary dramatically between individual lines.

Data summaries and their use in selection. Regardless of the breeding system, geneticists work from computerized summaries of data derived from test populations of individual birds. These may include calculated indexes including arbitrary economic weights, or estimates of income minus feed costs, or other economic parameters. The use of computerized summaries permits the concentration of large volumes of data for consideration. It also increases the complexity of the selection process. The use of indexes helps in eliminating individuals and families which would never be selected, but an element of judgment is almost always required in selecting birds or families close to the dividing line between selection and rejection.

The relative importance assigned to individual, full-sib and half-sib information in selection decisions varies considerably. Some breeders compute the indexes described by Osborne (1957a, 1957b) which give the optimal combination of these sources of information, based on genetic parameters within the population. Others apply more arbitrary weightings, based on experience with the population and the need to apply compromise to the selection process where dictated by circumstances. For example, where not all traits are included in the index, it may be necessary to reject some birds based on a type of independent culling level for traits such as shell quality, body weight, etc. These traits are rarely included in selection indexes, and are often dealt with by individual bird assessment. Thus, a member of a family with otherwise superior characteristics but with inferior shell quality might be rejected on these grounds.

Matings in pure lines. Matings in the pure lines are nominally at random, but with certain important restrictions. Most commercial geneticists attempt to minimize inbreeding, and therefore individuals with more than one grandparent in common are not usually mated. Within the relatively large populations that these breeders manage, this is not usually a major restriction on the randomness of mating. Another restriction would be that as far as possible the females within a particular mating group would be unrelated, at least in the immediately preceding generation.

The other restriction is that some breeders practice assortative or disassortative matings. Birds at the extremes of a selected range of body weights, for example, might be mated together to attempt to produce progeny of intermediate weight. This might also be practiced in the case of egg weight where females with otherwise unacceptable egg weights might be mated to a male whose sisters were at the other extreme for this trait.

Although inbreeding coefficients in commercial pure lines are seldom estimated accurately, and even more rarely reported, such values as do exist suggest a rate of increase below one percent per generation. In one population where detailed estimates were made, the inbreeding coefficients increased by 11.5 percent during 24 years of reciprocal recurrent selection (Ameli et al., 1988). Gowe and Fairfull (1980) reported that in two experimental lines selected for egg production traits for 29 and 27 generations actual inbreeding coefficients were 19.5 percent and 22.8 percent respectively. A control strain, randombred for 29 generations, had an inbreeding coefficient of 6.12 percent.

Experimental lines and test crosses. As well as the pure lines used to produce commercial crosses, most breeders carry a number of experimental lines at various stages of development. In many cases these are known to be suitable as substitutes for some of the main lines and many are so designated. This provides a measure of insurance against unexpected disasters affecting the pure lines, necessitating a rapid substitution. Most of these lines are held in fully pedigreed populations somewhat smaller than the commercial lines, and are under active selection. Such lines would be based on a minimum of 20 pedigreed sires. Selection goals may vary in experimental lines; they may be selected to meet anticipated future demands or to provide for a future substitution in the commercial cross based on prior knowledge of changed requirements.

The existence of both experimental and commercial lines implies that breeders also carry out large scale testing of potential new crosses. When breeders are striving to capture market share without an established product, literally hundreds of test crosses will be tested, representing most possible combinations of available pure lines. Once a superior product has been identified and established, the emphasis may change. As well as the identification of individual end products, the testing of crosses also highlights those pure lines with the best general combining ability. Subsequent test crosses will tend to concentrate on offspring from these lines at the expense of others with poorer combining ability, which may be dropped or amalgamated into new synthetic populations.

The test crossing procedure usually involves several hundreds of birds, frequently placed in a series of diverse environments. The object of this is, of course, to identify any genotype x environment interactions. In this context, the term environment must be used in its broadest sense. It would include the commercial environment and such factors as price differentials for different egg sizes, etc. While genotype x environment interactions, defined in the narrow sense, may not be frequent, breeders have sometimes experienced the problem of a product performing excellently in the country of origin yet being less acceptable on a wider basis.

Test crossing is usually carried to a much larger scale when only a few, or perhaps just one, test cross must be proved when exposed to commercial environments. At this point, breeders produce several thousand parents, and then distribute commercial-sized flocks alongside the existing product for comparative testing. Once these test birds become located outside the immediate control of the breeding company, the amount, precision and reliability of the data obtained are usually reduced. Nevertheless, with the aid of the breeder or hatchery service personnel, supervision of test flocks must be adequate to provide reliable data on which a final decision on a new product can be confidently based.

The use of commercial environments for testing of new varieties also permits the stocks to be exposed to multiple-bird cages and the increased level of disease usually associated with practical conditions. In fact, some breeders tested pullet families of pure lines in multiple-bird cages, to attempt to accomplish this, but the practice has largely been abandoned because it fails to provide records for individual birds. However, the use of multiple-bird cages for test flocks is important because it gives an indication of the behavioral qualities of potential new commercial stocks. Their ability to interact with each other and to survive in these more stressful environments is an important consideration. Increased disease exposure also permits breeders to obtain better estimates of general resistance to disease by measuring livability in the larger number of birds tested on commercial locations.

BREEDING FOR CHICKEN MEAT PRODUCTION

The development of the broiler chicken has resulted in birds with totally different properties from those used for egg production. This section will describe the selection criteria, the breeding and selection methods, and the strategies used in this segment of the industry.

The production of poultry meat, like that of table eggs, tended to be extremely diffuse and unorganized in its early stages. In most cases cockerel chicks, brothers to pullets grown for egg production, were used for meat production. This was particularly common in areas where brown eggs were produced; the cockerel chicks from such dual-purpose breeds were at least partly suitable for growth as meat birds. This situation prevailed in North America in the mid 1940s and in parts of Europe until the late 1950s.

The turning point for the fledgling broiler industry in the U.S. was probably the Chicken of Tomorrow contest sponsored by the Great Atlantic and Pacific Tea Company in

1946. This initiative, originating at the retail level, is an excellent example of the many stimuli which the broiler industry has received since that time. Most of the changes in emphasis which have taken place have resulted from changes in retail demand. This is in contrast to many other food producing industries which tend to receive most of their stimuli from the production, processing and marketing sectors.

Selection Criteria

The Chicken of Tomorrow contest focused sharply on several key aspects of consumer demand. These included an attractively shaped carcass, capable of being processed at weights demanded by the consumer, and available at a nonluxury price. Developments since that time have amplified these original demands and added to them a considerable number of others. Because of the structure of the broiler industry, it is convenient to divide the traits into those related to meat production, and those concerned with the economical production of baby chicks (Table 40.1).

Growth rate. The primary demand of broiler growers in attempting to satisfy market demand has always been rapid growth, with a maximum weight for age, or a minimum number of days to the desired market weight. Breeders have approached this target mainly by means of mass selection, using very high selection intensities based on body weight measurements at a fixed age. As the growth rate of stocks increased, the age at which measurements were taken was progressively reduced. Most evaluations for growth rate take place when the birds weigh between 1.5 and 2.0 kg. Very large populations were used, and, because the heritability for this trait is quite high, very rapid and consistent gains were made. Unfortunately, these gains were not without cost, particularly in terms of traits concerned with reproduction, and these consequences will be considered later in this chapter.

Feed efficiency. The rate of feed conversion, or feed efficiency, is another trait which broiler growers demand. During the time when selection for rapid growth resulted in such

Table 40.1. Selection criteria in breeding for chicken meat production.

Growth and production	Reproduction
Rate of growth	Age at sexual maturity
Weight for age	Rate of egg production
Days to market	Livability
Feed efficiency	Fertility
Body conformation	Hatchability
Meat yield and fat level	Color sexing of progeny
Yield of prime body parts	Feather sexing of progeny
Livability	
Absence of leg defects	
Absence of breast defects	
Rate of feathering	
Color of feathering	
Color of skin	

spectacular progress, the age at which broilers achieved the desired killing weight was progressively reduced. Under these circumstances, feed efficiency was also progressively improved, but in retrospect it is clear that this was almost entirely a consequence of the reduced amount of feed used for body maintenance during the shorter growing period. While weight for age is an easy characteristic to measure, especially in large populations grown under floor management conditions, the measurement of the feed conversion of individual birds is impossible under these circumstances. However, research studies have shown that individual feed efficiency varies among birds of the same weight (Fairfull and Chambers, 1984). This therefore offers another characteristic for which selection can be practiced, although one presenting major difficulties in measurement and interpretation. Breeders have approached selection for feed conversion in a variety of ways. The assessment of individual feed consumption during a relatively short test period is one approach. This may be restricted to males and/or to segments of the population which have already been preselected for rate of growth. An alternative method is to house family groups (full- or half-sibs) in community cages in which feed consumption can be measured. Marahrens and Flock (1980) reported a rather low heritability (0.14) from a two-year study of commercial populations housed by sire groups in cages.

Body conformation. One of the major differences between the broiler chicken and the cockerels from egg production stocks is in body conformation. The egg-type or dual-purpose bird is predominantly angular, while the meat-type bird presents a much more rounded appearance. This results from a combination of skeletal differences and muscle distribution. The use of Cornish Game birds and the development of specialized meat stocks from the Plymouth Rock and New Hampshire breeds was a direct response to the demand for a chicken with a meatier appearance. Within these stocks, considerable variation existed, and this was exploited by breeders in developing the modern broiler chicken. While a great deal of the breeding work has been based on subjective evaluations, such studies as have been conducted using objective measurements have indicated medium levels of heritability. The development of the breast angle metre permitted estimation of genetic parameters for breast angle, and some breeders have used the metre in modified form as a measurement tool.

While the breast angle metre may have had an impact, it is clear that to a large extent the conformation of modern broiler chickens is a testament to a relatively few skilled individuals who have been involved in subjective evaluation of broiler conformation over prolonged periods. Most breeders employ an individual or a small nucleus of highly skilled and experienced people who individually handle all of the pure-line birds, or at least all whose body weight justifies their selection as pure-line replacement breeding stock. The importance of these individuals to the breeding effort cannot be overestimated. Not only are they able to identify very subtle differences in body conformation but they are also highly skilled at detecting even minor defects and blemishes which defy measurement or identification by any other means.

Meat yield. While the initial selection for rate of growth is based mainly on live weight, the eventual success of the broiler operation is to a large extent dependent upon the yield of saleable meat resulting from the live chicken grown. Further refinements to this principle include the estimation of yield of prime cuts, especially breast meat, which command higher prices than some of the other components. Like feed conversion, yield is an extremely difficult characteristic to assess. In most cases, it can only be measured accurately after the bird has been slaughtered. In distinguishing between different breeds or lines when breeders are faced with such a choice, samples of each alternative may be grown out and processed, and yield measured. However, at the individual bird level the only way that yield can be approached in the breeding context is by the slaughter of sibs or progeny of birds available for selection. This is an expensive procedure, and any progress resulting from such selections is inevitably slowed down by the fact that the surviving birds can have no more than a 50 percent relationship with those which were evaluated. However, the carcass yield of a broiler is an extremely important attribute, and breeders must recognize this in assigning breeding priorities.

Fortunately, the yield or relative proportion of breast meat is positively related to some of the aspects of conformation which were described previously. Thus, birds which were developed to meet high conformation standards tend to have acceptable yields of breast meat. Even here, individual evaluation of sibs may provide further possibilities for selection in the pure breeding lines.

Mortality and condemnations. The broiler growing industry is extremely sensitive to losses resulting from mortality, condemnations, and carcass defects. Mortality may result from exposure to disease and breeders of broilers have attempted to minimize disease in much the same way as the breeders of egg-type stocks. Many congenital diseases have been eradicated. Some selection for resistance to certain diseases, for example lymphoid leukosis, has been imposed, and appropriate vaccination programs have been developed for other diseases. However, some of the death loss among broilers seems to relate to their extremely rapid growth. While no direct evidence of genetic susceptibility exists, the possibility of such a relationship is not overlooked by broiler breeders. Some of the mortality may be self-limiting, since it appears to be restricted to the most rapidly growing individuals in the population. Any other genetic approach to reducing mortality is made extremely difficult by the relatively low incidence; among commercial broilers, losses in excess of four percent are uncommon, and in pure lines maintained by breeders the level of loss may be much less than this.

Leg defects. Broilers which survive to slaughter age but exhibit physical imperfections may be totally condemned or may have a much reduced value resulting from the defect. Perhaps the most common of these faults are those affecting the legs. Many of these become exaggerated as the birds grow older and achieve high body weight. At least some of them can be detected in their early stages by skilled observers, long before they have a visible or harmful effect upon the growing bird. Although leg defects are usually combined as a general category in assessment programs, they are anatomically distinct, and where genetic influences exist they may be assumed to act independently. The most important specific deformities include:

Tibial dyschondroplasia in which legs appear bowed due to abnormal development of cartilage in the end plates of long bones. It is most common in the proximal growth plate of the tibia. Both genetic and nutritional influences are involved in the expression of tibial dyschondroplasia.

Slipped tendon which is called perosis when it results from a manganese deficiency. It may occur without any known nutritional deficiency, and is characterized by the gastrocnemius tendon slipping off the condyles.

Chondrodystrophy in which long bones are unusually shortened and/or distorted. While major genes have been identified which cause extreme and predictable incidence of chondrodystrophy, less severe forms appear at low frequencies.

Spondylolisthesis (kinky back) which is a dislocation of the sixth thoracic vertebra with overriding by the seventh. This causes spinal cord pinching and vertebral cartilage defects. It is not really related to leg problems, but affected birds have difficulty walking, which results in its inclusion in the general category.

Angular bone or valgus-varus deformity involves a lateral or medial angulation of the shaft of the distal tibiotarsal bone (Julian, 1984). This results in deviation of the lower part of the leg and, frequently, the bending of the proximal shaft of the tarsometatarus.

A variety of other leg disorders may occur, but they result from specific nutritional or mechanical circumstances. They are therefore unlikely to form part of a selection program. Selection against many of these leg defects is made extremely difficult by the fact that pure-line populations seldom exhibit them at a level which would make genetic progress through selection a possibility. Clearly, all birds which appear deformed at any stage are rejected from breeding programs. Yet commercial broilers continue to exhibit the defects. This emphasizes the point that many leg deformities result from the interaction of genetic predisposition with the highly intensive management and disease status of commercial flocks. By artificially increasing the incidence of leg problems through diet and high density management, and subsequent selection, some breeders have brought about genetic improvement. Great reliance is also placed upon physical examination and identification of early symptoms in individual birds with an anatomical predisposition to subsequent leg weakness.

Breast defects. Another source of loss in commercial broilers is the so-called breast blister. This is manifested as damage to the skin covering the keel bone on the finished broiler. It is highly sensitive to environmental conditions; insufficient or wet litter tends to aggravate the condition and increase its incidence.

However, another important factor is the shape of the skeleton, particularly the relationship of the keel to the spinal column. Gristwood (1988, personal communication) has provided graphic demonstration of the potential of contrasting skeletal types to develop breast blisters. In the accompanying diagrams (Figure 40.2) the A type represents the undesirable skeleton. In these birds the keel and the spinal column are essentially parallel; the front end of the keel projects beyond the clavicle and can be clearly seen through the breast muscles. When these birds sit down, the body weight rests mainly on the point of the keel, with a corresponding high risk of breast blisters. In the contrasting B type the keel is at an angle to the spinal column creating a wedge shape. The line of the clavicle merges smoothly with the line of the keel which does not protrude through the breast muscles. When these birds sit, body weight is evenly distributed along the whole keel, minimizing the risk of breast blisters. Breeders have progressively modified their lines so that most now conform to this desirable type, which also results in improved carcass appearance. As with reductions in leg problems, this has been accomplished entirely by skillful observation and handling by experienced selection crews.

Feathering on the breast also plays a part in protecting the bird from breast blisters. Thus, in their physical examination of pure line stocks at the time of the initial selection, breeders examine birds for skeletal shape, feather coverage, and early symptoms of the development of breast blisters. Individuals not meeting standards are rejected.

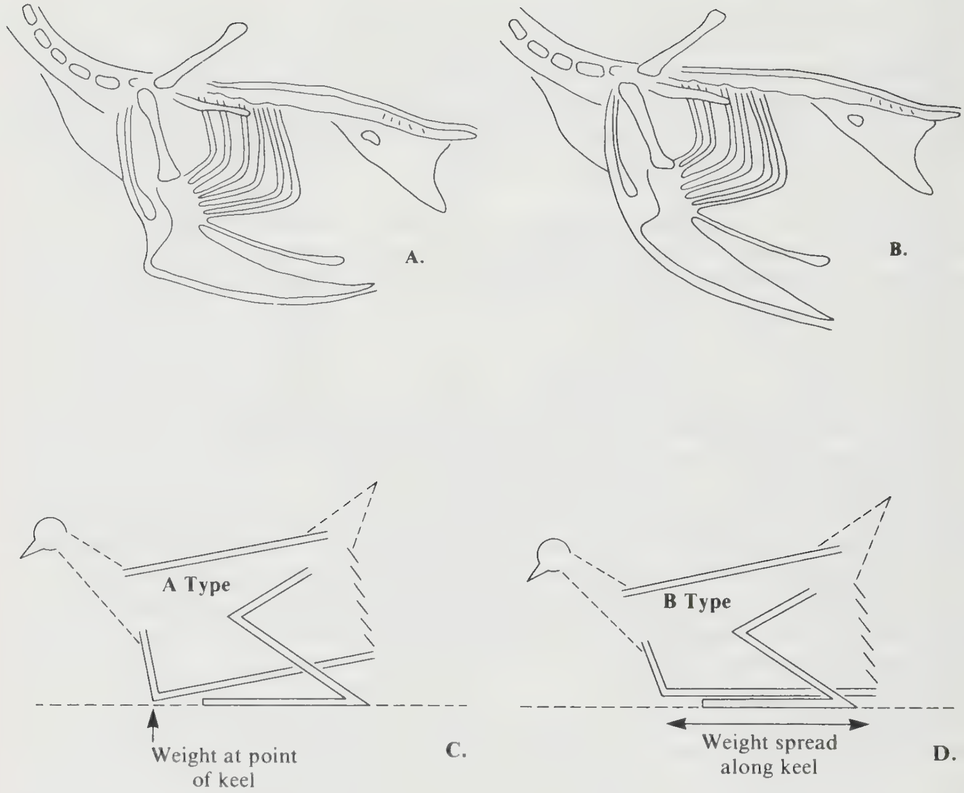

Figure 40.2. Contrasted skeletal conformation with (A) pointed keel having a predisposition to breast blisters and (B) smooth rounded keel with greater resistance to breast blisters and improved appearance. When sitting on the floor, broilers with these skeletons assume positions shown in (C) where all the weight is concentrated on the point of keel or (D) where weight is distributed along its length. (Courtesy Alan F. Gristwood).

Appetite and body fat. Genetic changes resulting from long-term intense selection for body weight may have provoked some of the problems related to leg weakness and breast blisters referred to above. It also resulted in chickens with greatly increased appetites. In fact some experienced observers believe this to have been the most important consequence of the early selection procedures, even though appetite itself is not directly measured. Nevertheless, it is clear that the broiler chicken has a very heavy demand for feed. The ability of the bird's metabolism to transform feed into edible meat was accepted for a long time without question. More recently, the partition of the edible meat into protein and fat has received considerable attention. The presence of relatively large amounts of body fat in broiler carcasses began to appear as a problem during the 1970s. Consumers purchasing whole birds or parts were confronted with quantities of abdominal or leaf fat which was generally trimmed and discarded prior to cooking. As the industry moved towards further processing and individual parts or components of the chicken carcass were sold separately, the excess body fat became a loss to the processor since none of the customers would accept it. While it is evident that the amount of carcass fat is dependent to a large extent upon nutrition, it is also partly under genetic control. It is quite clear from the work reviewed by Leenstra (1986) that genetic reductions in total fat content are quite feasible.

Leenstra et al. (1986) and Leenstra and Pit (1987; 1988a, 1988b, 1988c) provided experimental evidence that fat deposition in broiler stocks responds to selection. The problem faced by broiler breeders is the measurement technique. The measurement of total or abdominal fat involves the slaughter of the subject bird, in the same way as is necessary for direct measurements of carcass and parts yield. Therefore, breeders have sought to discover alternative measurements on live birds which could be used to predict levels of abdominal fat. These methods have included physical palpation of the abdominal region, and measuring wing web thickness and the thickness of a pinch of skin at the centre of the humeral feather tract (Mirosh et al., 1981). Pym and Thompson (1980) developed a caliper which was shown to be effective as a tool for estimating abdominal fat, but others have not always been able to repeat their results in practical conditions with less diverse source material.

Griffin and Whitehead (1982) proposed the use of blood levels of very low density lipoprotein (VLDL) triglyceride concentration as a selection criterion. The VLDL measurement has been shown to have a high correlation with total body fat as well as abdominal fat. While this method involves a relatively complicated assay, it can be used on young birds and has been adopted by several breeders.

Another approach to reducing body fat levels has been to exploit the relationship between feed efficiency and body fat deposition. Both commercial observation and published research (see review by Leenstra, 1986) indicated that as feed efficiency is improved, the level of abdominal fat diminishes. Therefore, breeders selecting for improved feed efficiency anticipate a correlated downward response in abdominal fat level. While these correlations are not high, they are in a favorable direction for the breeder. Furthermore, some breeders believe that excessive reductions in abdominal fat would be counterproductive since they might be accompanied by undesirable changes in other characteristics such as succulence and flavor which depend upon fat for their expression.

Reproductive fitness. Many of the meat traits have relatively high levels of heritability. This is clear from published research, industrial observation, and breeders' own records. Rapid progress, especially in growth rate for age, became an easily achievable goal for broiler breeders. Using mass selection only, progress in the order of 50-100 g per generation at a fixed age, or a progressive reduction in slaughter age of 1-2 days per generation, were commonplace.

However, almost from its beginning the nature of the broiler industry was such that although these improvements were welcomed, the associated deterioration in reproductive performance was not. Most broilers are grown by integrated operations which not only oversee the growing and processing of meat birds but also own or manage the breeding flocks required to produce baby chicks. A range of reproductive characteristics related to broiler chick production must also be included in the primary breeders' repertoire. Some of the traits are similar to those under selection in egg production stocks while others are somewhat different. It must also be emphasized that a great deal of the success or otherwise of commercial broiler breeding operations is attributable to nongenetic factors such as nutrition and lighting. Breeders tended to exploit these before seriously tackling the problems of reproductive performance at the genetic level.

The breeders' first approach to genetic improvement of egg production is to identify pure lines and crosses which are superior for this characteristic. Since egg production is only required in the female parent, this was accomplished by identifying lines which were originally derived from dual-purpose stocks such as White and Barred Plymouth Rocks, New Hampshires, etc.

Measurement of reproductive efficiency begins with trapnesting to determine *age at maturity* and *rate of egg production*, in much the same way as is practiced in egg-type breeding lines.

Some breeders, in addition, study *fertility* in both male and female lines and embryo viability or *hatchability* in female lines. Because the cost of producing broiler hatching eggs is comparatively high, the ability of an embryo to hatch acquires proportionately greater importance, compared with the cheaper egg-type embryo.

In contrast to the meat traits, the reproductive characteristics are sex-limited and have low heritability levels. This creates added complexity both in the identification of goals and the measurement of characteristics aimed at achieving them. Adding further to the complexity is the fact that in general the meat traits and reproductive traits are negatively correlated at the genetic level. A variety of genetic and nongenetic techniques has been employed to address these problems. There is an obvious opportunity to trade off one set of characteristics against the other and breeders have used various strategies in attempting to optimize their total selection programs.

Balancing growth vs. reproduction. The weights assigned to growth and reproductive traits in broiler selection programs are of major concern to commercial breeders. While breeding companies are well aware of the theoretical methods available to optimize the weights applied to various traits, they are also influenced by market forces which are not taken into account by these models.

Moav and Moav (1966) were among the early contributors to this field. An important conclusion from their work was that when the initial reproductive level was higher, the change in profit resulting from a unit change in reproductive performance was correspondingly lower. Hunton (1969) also demonstrated the relatively small effect on cost of production of genetic improvements in egg production, when compared with the savings which could be expected from genetic gain in weight for age. Abundant field evidence also attests to the value of improving broiler production traits like growth, feed efficiency, and carcass yield in contrast to breeder characteristics like egg production or hatchability.

The relatively simplistic nature of the above studies is highlighted by the complexity of the models presented by Harris et al. (1985) and Akbar et al. (1986). The application of these systems to commercial broiler breeding, however, ignores two important features of the industry: firstly the fact that breeders have a wide range of customers whose needs vary across a broad spectrum, and secondly that priorities change quickly due to the dynamic nature of broiler marketing.

In practice, the determination of weightings for reproductive versus growth traits is most likely accomplished by a combination of relatively few factors. Of primary importance is a company's position in the total market. A dominant breeder can probably afford the luxury of choice. Strategy may be at least partly determined by calculation of a rational optimum for weighting various traits. On the other hand, contenders with only minor market share *must* be competitive with the market leaders for the major growth and reproductive traits in order to survive.

Some breeders identify a particular market segment and aim selection policies specifically to meet its requirements. An example might be broiler hatcheries who are not part of integrated corporations and are, therefore, profit centres in their own right. Such customers place high priority on parent performance and efficiency, fertility and hatchability. They are concerned with the cost of producing a broiler chick, not with that of producing a kilogram of chicken meat as is the case with the integrated organization.

Other broiler companies may require added emphasis to finished carcass appearance. Where a significant proportion of output is sold in branded form this may be an important selling point. Conversely, where most output is further processed, cut up, or sold in commodity form and unidentified, the priority would be low cost per unit of product.

Selection Methods Used By Meat Chicken Breeders

Because the primary traits of rate of growth or body weight at a fixed age have quite high heritability, much of the early progress in meat stocks resulted from mass selection. The size of the industry which bought the breeding stock, and its rate of expansion, made it possible to select from huge populations. Because demand for parent stock, and thus grandparents, was continuous, pure lines could be hatched every week with overlapping generations. This permitted immediate incorporation of selected generations into the commercial program. Very high selection intensities were made possible, resulting in extremely rapid gains. Initial selection intensities retaining approximately one percent of males and ten percent of females were quite common. Because even such highly selected stocks had a reasonable rate of reproduction, these intensities could be maintained for several generations without seriously eroding egg production, hatchability, etc. Some progeny testing work was also included, but the main breeding technique seems to have been relatively simple mass selection.

In the United States, where most of the initial selection work on meat stocks took place, breeders tended to specialize in either male or female stocks. Thus, a number of breeders emerged selling male line stocks mainly derived from the Cornish breed, and others selling female lines originating from Barred Plymouth Rocks, White Plymouth Rocks, New Hampshires and other originally dual-purpose breeds. This was beneficial to the commercial industry in several ways. It ensured that any benefits from general combining ability of the male and female stocks used would be exploited. While male breeders may have relied initially on a single line, female breeders whose product was sold at much lower prices relied on two-way crosses to a large extent. This meant that there was some heterosis at the grandparent level and good egg production and hatchability could be anticipated from the resulting two-way cross parent females. Broiler growers were also assured of a commercial chick with characteristics intermediate between very heavily selected meat type male lines and female lines with at least acceptable growth rate characteristics.

Selection within the female lines has always included early assessment of growth and conformation, followed by varying levels of attention to egg production, fertility, and hatchability. Because these traits are sex-limited and have low heritability, populations have had to be fully pedigreed or at least pedigreed to sire to permit any effective selection and improvement. Many breeders only initiated pedigree breeding and trapnesting after many generations of mass selection for body weight and conformation.

As the industry developed and competition intensified, most breeders began to develop both male and female lines. The advantage of this to the eventual customer was a reduction in the risk of health problems resulting from placing chicks from two different origins on the same farms. Additionally, any specific combining ability which existed between the male and female lines could be exploited. The use of two-way crosses was often extended to the male line stocks, both to protect the breeders' germplasm and to exploit any heterosis which might lead to greater economy in producing male parents.

As emphasis began to be placed on traits like body fat, feed efficiency, and other traits with lower heritability than growth rate, full or part pedigreeing of all lines has become universal. While adding significantly to cost, this procedure also provides a much more complete assembly of information with which the geneticist can work to obtain optimum progress.

The task of the broiler geneticist has thus become in some respects even more complex than that of the professional working with egg-type stocks. Data on a wider range of traits gathered over a much longer period must be combined and analyzed. Known negative correlations between traits of primary importance can seriously hamper overall progress. And the rapid evolution of the commercial industry ensures that new demands on breeders are made each generation.

Monogenic Traits In Meat-Type Chickens

Early in the development of the broiler industry, the market began to demand exclusively white-feathered birds. This was mainly to achieve uniformity of appearance; a few colored pinfeathers could easily mar the appearance of an otherwise acceptable carcass. White pinfeathers were not so obvious. Thus, most of the broiler strains sold carry the gene for dominant white, I, at least in the male line. This gene completely inhibits the development of black color, although it does not prevent the development of variable amounts of red coloring.

A few small but significant markets exist for meat chickens with colored plumage. In parts of Europe, notably in France, specialist growers produce extensively grown meat birds with colored (usually red or black) plumage. These are slower growing strains than the conventional broiler, and appeal to gourmet markets. In parts of Asia, white birds are unpopular because of religious beliefs, and red-feathered strains have been developed to replace them. In many cases, the birds are marketed live, and the problem of pinfeathers spoiling the appearance of mechanically defeathered carcasses does not arise.

Regional preferences for skin color have occasionally encouraged breeders to use the autosomal genes W^+ and w to provide white- and yellow-skinned broilers respectively. The W^+ gene prevents the deposition of yellow pigments derived from the diet in the skin, fat, shanks and beak. It is completely dominant to the w gene, which permits the deposition of the pigments. However, birds homozygous for the w gene must be fed adequate amounts of such pigments in order to express the yellow-skinned trait. Most commercial broilers are w/w, and the degree of yellowness in the skin is determined by nutrition; those growers requiring highly pigmented birds feed high-corn diets, sometimes supplemented with natural or synthetic xanthophylls, while those requiring lower levels or no pigmentation use diets based on milo, wheat or other cereal sources lacking the pigments or their precursors.

Because of sexual dimorphism with respect to some growth and carcass characteristics (Figure 40.3), some broiler producers require separation of the sexes in order to serve different markets. Sexual dimorphism increases with age; at 28 days females weigh 85-88 percent of males while at 56 days they weigh only 82-83 percent as much.

Both *feather sexing* and *color sexing* have been applied to broiler stocks. The sex-linked gene for slow feathering (K) has been incorporated into female parent lines. Thus, when mated with the homozygous fast feathering (k^+/k^+) male, the male broiler chicks are slow feathering (K/k^+) while the females are fast feathering ($k^+/-$). The distinction between the sexes can be easily made in the hatchery, and does not require the specialized labor needed for vent sexing by the Japanese method. The slow rate of feathering of the male

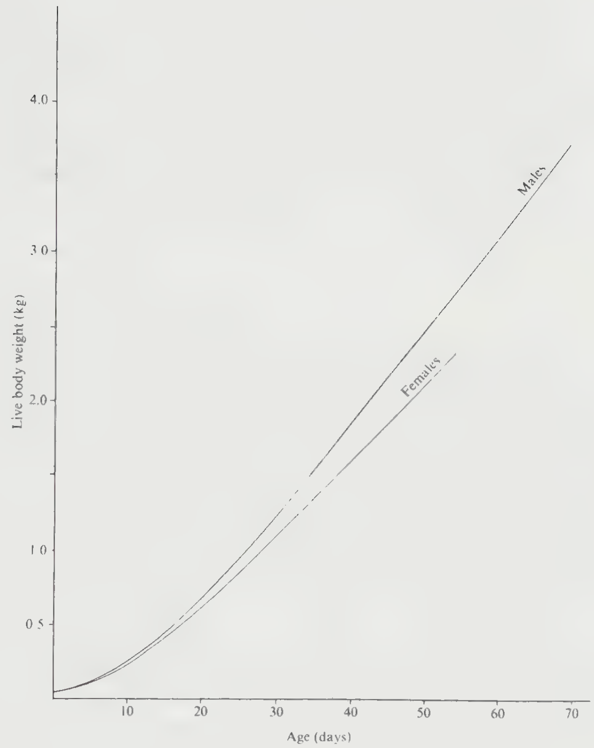

Figure 40.3. Growth rate of male and female chicken broilers. (Courtesy Cobb Breeding Co., 1987).

broiler chicks has occasionally created problems, but the rate of feathering improves as the birds grow older, so only where males were killed very young did they suffer disadvantages conferred by their *K* gene.

The use of the silver/gold (S,s^+) locus to provide color sexing in broilers is an alternative to feather sexing. However, this is complicated by the requirement for some of the other genes determining plumage color. The broilers are still required to be dominant white (I/I or I/i^+). In addition, it is necessary for both sexes to be homozygous for the columbian pattern, permitting the gold down to be observed in baby chicks and red coloration on the adults. When these conditions are met, color sexing allows easy sex differentiation without the potential disadvantages of the slow feathering gene.

The sex-linked gene for dwarfism (*dw*) described by Hutt (1949) has been incorporated into some broiler female parents as suggested by Jaap (1969). This reduces the body size, space, and feed requirement of the parent female by 20-30 percent, thereby increasing the efficiency of chick production. Where this gene is used, it is normally incorporated in the line which will be the male line in the grandparent mating to produce female parent stock. In a four-way broiler cross (AxB) x (CxD), the dwarf would be in the C position. This means that it is possible to use the same D line for the production of both normal size and dwarf parent females. The A and B lines could also be the same in the normal and dwarf systems. In the case of the dwarf parent, all the commercial progeny will be normal size but the males will be heterozygous (Dw^+/dw). In many cases, due to a combination of slightly lower egg weight and incomplete dominance of the Dw^+ gene, a reduction in growth rate of males of up to three percent may be observed. This sometimes limits the application and usefulness of the dwarf gene, but in many circumstances dwarf female parents provide an economic advantage.

In the case of the *dw* gene, as with all other desirable single genes, the commercial user demands its incorporation without sacrificing existing genetic characteristics. This inevitably slows down the introduction of varieties possessing these exotic genes.

BREEDING AND SELECTION OF TURKEYS

The commercial exploitation of turkeys is not nearly so widespread as that of chickens. Although the turkey is a native North American bird, today's commercial varieties bear little resemblance to the American wild turkey. Most of today's turkeys are originally descended from the Broad-breasted Bronze, which itself was derived from a cross between turkeys developed in England and the American Bronze. The latter was a hybrid of the original domesticate and the wild subspecies of eastern North America. Because the turkey industry is limited to relatively few countries, very few companies are involved in turkey breeding. In fact, in 1988 only three companies made a significant contribution to the world's supply of turkey breeding stock. In addition, a few independent companies bred turkeys for their own needs.

Mature turkeys vary considerably in size, and during the development of the turkey industry this fact was put to good use. Turkeys were developed for slaughtering at several different ages for different market purposes. For example, individual consumers require turkeys in the 5-10 kg range, and these could be supplied either by mixed sexes from some of the smaller types or from the females of the larger varieties.

Sexual dimorphism in meat turkeys has important influences on rate of growth and fat deposition. Typical growth rates for one commercial heavy turkey variety are given in Figure 40.4. Fat deposition begins earlier and is more pronounced in females.

Turkeys sold as whole carcasses require subcutaneous and intramuscular fat to provide both visual and organoleptic appeal. While this is influenced to some extent by feeding programs, immature turkeys, especially males of some strains, do not possess adequate fat cover for market requirements. For this reason hen turkeys are favored for killing and marketing as whole carcasses. Males of the same strain are carried on to higher weights for use in markets where whole carcass appearance is not important, e.g. deboning and/or further processing. In addition, strains have been developed which mature at a relatively young age and at modest weights to meet whole carcass demands. Such turkeys would not be suitable for the catering or further processing segment of the market. Small or broiler turkeys also experienced a period of relative popularity, leading to the development of the Beltsville Small White.

However, as the breeding industry became concentrated in relatively few hands, the availability of some of these different types diminished. Most of the growth in the turkey industry is now in the area of large turkeys killed at the highest weight possible, and then cut up, deboned, further processed and/or reconstituted into new forms. This has led to a concentration on the rapidly growing turkey at the expense of the smaller varieties. Nevertheless, some breeders can offer a range of turkeys to meet different demands.

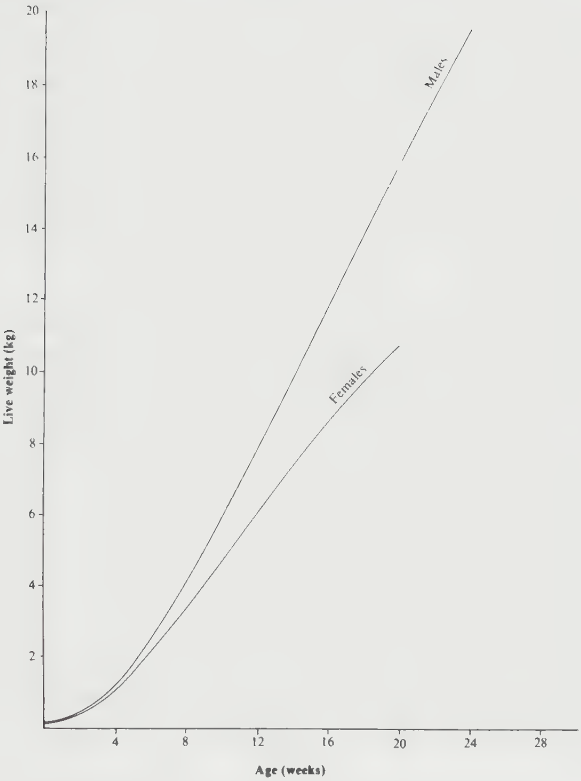

Figure 40.4. Growth rate of male and female turkeys. (Courtesy British United Turkeys, 1986).

Selection Criteria

Modern turkey strains have been selected almost exclusively for traits related to growth and conformation. Reproductive efficiency has provided a major challenge in the commercial turkey, since the development of fast-growing broad-breasted strains produced birds which were unable to mate naturally with any degree of efficiency.

Growth and conformation. Growth rate or weight for age in turkeys has been shown to have a similar heritability to that in chickens. Thus, selection for these traits has been highly effective. In fact, the modern turkey is one of the most rapidly growing species in commercial poultry farming (Figure 40.4). Along with growth rate, improved body conformation has also been a major object of selection. A comparison of the turkey of today with the traditional wild turkey reveals a similar picture to the one seen when broiler chickens are compared with Leghorns. The attention to breast conformation has resulted in the so-called dimple-breasted appearance, with extremely heavy musculature in the breast region. While mechanical means may exist to measure this characteristic, most breeders rely on visual assessment of individuals for breast width and length.

Growing turkeys also suffer from various leg defects and weaknesses. These are frequently aggravated by environmental conditions, particularly relating to the floor litter. As in broiler chickens, some of the leg weaknesses may be partly inherited, and individuals with leg defects are therefore rejected in breeding programs. It appears that the fastest growing turkeys are the most susceptible to leg problems, and therefore these strains, particularly the males, are subjected to rigorous examination during their growth period. In addition to examination for individual defects of the skeleton, the stance of the live bird is also considered with a view to placing the centre of gravity of the entire body directly over the legs (Figure 40.5). This necessitates observing the birds standing and walking unhindered, as well as while restrained for determination of conformation.

Reproduction. The turkey industry is probably unique in that virtually all commercial matings are undertaken using artificial insemination. The development of A.I. in turkeys occurred at a fortuitous time; following the demonstration of A.I. in chickens by Burrows and Quinn (1935), the same workers (1937) reported the successful collection of semen from turkeys. Two decades later, when broad-breasted turkeys emerged, the technique was invaluable for improving fertility. The excessive weight, breast width, and poor balance of these birds made natural mating difficult if not impossible. Very quickly all commercial turkey reproduction changed to A.I. While this enabled these strains to reproduce with reasonable efficiency, it did nothing to arrest the gradual decline in reproductive performance accompanying rapid selection for growth and conformation. Thus, the reproductive ability of the large strains of turkey leaves much to be desired. Selection for egg production receives significant but limited attention from turkey breeders. One major contributor to variation in reproductive efficiency is broodiness. Selection against broodiness has been an important component of some breeding programs. While among commercial turkey producers efforts may be made to break the broodiness cycle and return the females to egg production, in breeding programs birds which become broody are usually eliminated.

Breeding And Selection Systems For Turkeys

Commercial turkeys are usually three-way or four-way crosses, following the same pattern as in other commercial poultry. Pure lines destined to contribute to the male side

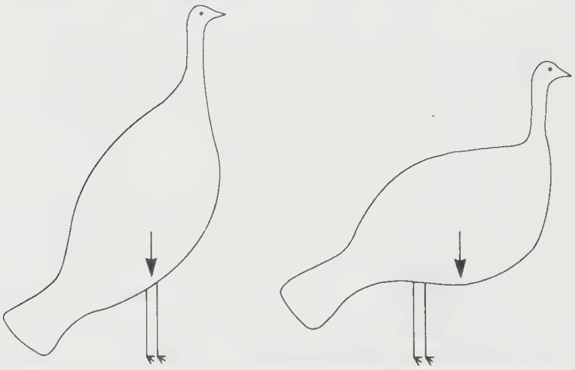

Figure 40.5. Profiles of contrasting body stance in turkeys. Arrows indicate centre of gravity; over legs (left) is desirable, over keel (right) is undesirable. (Courtesy British United Turkeys, 1986).

are frequently selected almost exclusively for growth and meat-type traits. They may be held in pedigreed populations; more commonly they are pedigreed to sire only. Population sizes are usually large, with intense selection. While the primary selection intensity is based on measurements of weight gain, etc., there is a tendency to place considerable emphasis upon visual and tactile assessment by expert handlers. Characteristics such as muscle development, carcass conformation, absence of physical defects including leg weaknesses, and correct stance are maintained or improved as a result of this examination. The birds will be handled several times prior to being used as pure-line replacement stock. Initial assessments are made at the earliest likely killing age; a majority of birds will be rejected at this time. Those remaining will be assessed again at least twice before reaching sexual maturity. Selection intensities practiced by breeding companies are not divulged; nevertheless it might be anticipated that in male lines they may approach the intensities used for meat chickens.

Lines destined to form part of the female side of the turkey breeding package are mostly held in fully pedigreed populations, although some breeders still pedigree only to sire. In this case, the male will be mated with several females, but the eggs will only be identified to the male parent and progeny will be a mixture of full-sibs and half-sibs. Selection for growth and conformation forms an important part of the selection process for these lines, but intensities are not as high as those practiced in the male lines. All birds are evaluated for growth and conformation at approximate market age, but relatively large populations are retained to test reproductive characteristics. Although some attempts have been made to manage breeding female turkeys in cages making egg production recording easy, most populations undergoing selection are now held in floor pens with trapnests used to obtain individual egg production records. The larger strains of turkeys are almost impossible to maintain in cages due to the size of cage required and the risk of damage to the turkey's feet. In addition, the cage environment tends to suppress broodiness and thus the identification of potentially broody females is not possible. In addition to egg production records, the use of trapnests enables pedigree identification of eggs and possible evaluation of fertility and hatchability of both males and females.

Although genetic progress may result from individual and family selection for egg production records, the main method of improving turkey reproduction at the commercial level is the careful exploitation of heterosis. The identification of two-way cross females with superior reproductive performance again serves the dual purpose of achieving acceptable rates of reproduction along with protecting the breeder's germplasm from possible use by competitors.

Most turkey breeders have a number of unrelated pure lines under selection. However, their policy in terms of commercial stock offered for sale varies. Some breeders offer only a single genetic combination to serve all markets. Others provide a range of varieties bred to achieve small, medium, and large mature weights. An alternative is to combine pure lines on a custom basis for individual markets. Thus, the products of the various commercial breeders vary considerably even though they attempt to serve the same range of customers. Another source of variation among commercial turkeys is thought to be the result of slightly different emphasis placed by the selection teams of the different breeders in assessing conformation in individual pure-line birds. Breeders believe that distinctive carcass and body shapes have resulted from this source of variation.

BREEDING DUCKS AND GEESE

Ducks and geese are grown commercially in various parts of the world, but on a much smaller scale than either chickens or turkeys. The main market for commercially raised ducks and geese is for meat. Duck egg consumption is significant in a few countries, particularly in Asia where in some cases it exceeds the consumption of chicken eggs. In addition, the feathers and down of ducks and geese have a considerable commercial value, being harvested either from the live animal or more commonly at the time of slaughter.

Domestic And Muscovy Ducks

Both the domestic duck (*Anas platyrhynchos*) and the muscovy duck (*Cairina moschata*) are grown commercially. Representatives of the two species can be crossed to produce a sterile hybrid, commonly known as the mule duck or mulard, which is also grown commercially for meat in several parts of the world.

There are significant differences between the species. The growth patterns and sexual dimorphism are illustrated in Figure 40.6. Sexual dimorphism is a major factor in the muscovy duck, males being about 75 percent heavier than females, while in the domestic

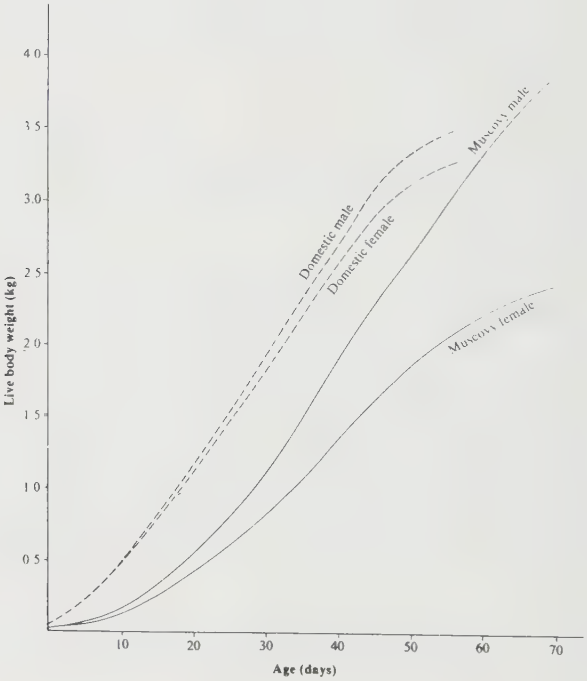

Figure 40.6. Growth rate of domestic and muscovy ducks. (Courtesy Cherry Valley Farms Ltd., and Grimaud Frères, S.A., 1988).

duck, male weights exceed females by only about six percent. The muscovy develops limited body fat deposits which decline after eight weeks of age, while the domestic duck is characterized by higher fat levels which increase with age. Egg production in the muscovy is comparatively low, while in some varieties of domestic duck over 300 eggs per year may be expected. The mule duck is intermediate between its two parents in growth and fat deposition.

The relatively small size of the duck industry has resulted in proportionately fewer commercial breeders working with these species. In 1988 there was just one major breeder of the domestic duck and one of the muscovy duck. In addition, a number of smaller breeders, mainly supplying only their own needs, were active. Comparatively little research has been carried out with ducks at the institutional level; therefore, breeders and geneticists have had to assume that data from chickens and turkeys could be extrapolated to ducks.

It is fairly evident that both species of ducks will respond to selection for improved growth rate or weight for age. Market demand for increased body weight has not been as influential in the duck industry as with chickens and turkeys. In addition, ducklings are usually marketed at a specific age immediately prior to the second molt in order to avoid having pinfeathers on the carcass. Because of this, attention has been paid to another important aspect of meat production, feed conversion efficiency. Powell (1986) described the results of nine generations of selection for improved feed conversion efficiency, using individual measurements of feed intake. He reported the results shown in Table 40.2.

This is one of very few commercial selection programs which was conducted in the presence of an unselected control. Therefore there can be considerable confidence that real genetic change was accomplished. Furthermore, these data show that not only were the ducks more efficient following selection, but also that the levels of body fat were significantly reduced. While this has been the objective in commercial selection efforts with other species, it has never been so convincingly documented as in the case of these duck populations.

Reproduction in meat-type ducks has never been as efficient as in the breeds developed for egg production, for example the Khaki Campbell. Improvements have mainly been achieved by selecting between different strain crosses in contrast to within-line selection. The international breeders therefore use strain crosses for their meat ducks, although some independent breeders producing for their own use continue to use pure lines. Improvement of management and environment has also been used to raise the egg production of meat-type parent ducks to acceptable levels.

Table 40.2. The results of nine generations of selection for improved feed conversion ratio in three different strains compared with their unselected control populations. (Modified from Powell, 1986).

Strain	Age at market, days	Liveweight, g	Feed conversion ratio	Skin and subcutaneous fat, %
A Control	49	3563	2.61:1	38.87
A Selected	49	+369	-0.27:1	-4.59
B Control	48	2727	2.98:1	33.33
B Selected	48	+417	-0.47:1	-5.01
C Control	48	2195	3.14:1	32.78
C Selected	48	+495	-0.62:1	-6.64

Geese

Goose breeding has also received comparatively little attention from geneticists and breeders due to the small total market. In addition, the goose has traditionally been regarded as having a high incidence of carcass fat, which, in the context of consumers' desire to limit fat intake, is a negative perception. A number of relatively small organizations breed geese commercially, mainly to supply their own needs for the production of goose meat and sometimes for down feathers. There is no world-wide distribution of goose breeding stock comparable with that for chickens or turkeys. Because reproductive rates are relatively low, the cost of producing goslings is high, and even though geese are capable of scavenging and living off low-cost feeds and even pasture, the total cost of production is such as to place the goose in a luxury category. Where markets exist, these are mainly for the sale of whole birds which also limits the acceptance by consumers.

Considerable basic research will be required in the areas of management, reproduction and nutrition if the goose is to compete with other poultry and other meats.

Where breeders have selected for improved growth rate or weight for age, responses have been reported. Some breeds of geese have a very rapid rate of growth and this can be exploited in commercial circumstances.

REPRODUCTION AND DISTRIBUTION OF BREEDING STOCK

Breeders are faced with a significant problem in allocating resources to research, multiplication, and experimentation. All have a limited amount of physical resources and competing demands on them. For example, a breeder may have to decide between expanding a selection program with accompanying improvements in ultimate performance, and allocating housing to grandparent stock with immediate increases in parent stock sales. The total complex of activities is illustrated in Figure 40.7.

Another problem for breeders is ensuring that the birds sold at the commercial level are the same genotype as those identified as superior at the research and development level. Such factors as genetic drift or deterioration, or incorrect identification are real hazards which breeders strive to overcome. At all levels, physical identification of birds presents a constant challenge. Since pedigree breeding and reproduction first began, identification of individual birds has been by means of printed metal or plastic wing bands. These have the advantage that they can be placed upon day-old chicks, and later replaced or supplemented by other means of identification. The use of numerically sequenced leg bands or cage identification can be replaced by electronic identification resulting in much improved speed and accuracy in identifying adult birds. Nonpedigreed generations of pure lines held by breeders are normally identified by means of sequenced wing bands or occasionally by toe punching or clipping.

Once breeding stocks are sold, either at the grandparent or the parent generation, responsibility for the identification passes to the customer. Normally customers for parent stock receive only males of one line and females of another. At the grandparent level they may receive males of two lines and females of two others, which would require careful separation and identification.

With traits having high heritabilities such as growth rate, there is an opportunity for further selection and improvement between the pure line and the commercial generations. Broiler and turkey breeders would normally carry out mass selection at great-grandparent and sometimes at grandparent level prior to the production of parent stock for sale. Breeders

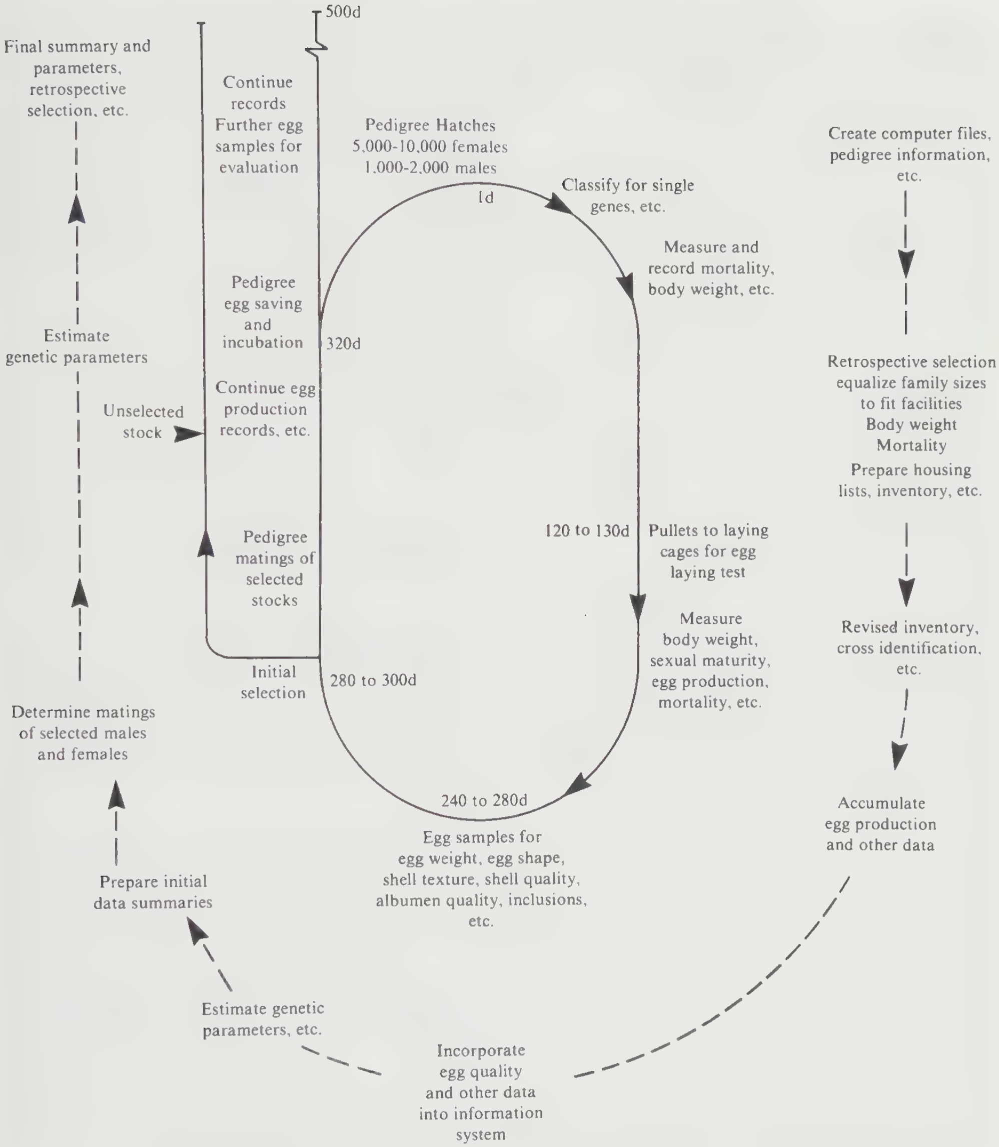
500d
Final summary and parameters, retrospective selection, etc.
Continue records
Further egg samples for evaluation
Pedigree Hatches
5,000-10,000 females
1,000-2,000 males
1d
Classify for single genes, etc.
Create computer files, pedigree information, etc.
Measure and record mortality, body weight, etc.
Pedigree egg saving and incubation
320d
Estimate genetic parameters
Continue egg production records, etc.
Unselected stock
Retrospective selection equalize family sizes to fit facilities
Body weight
Mortality
Prepare housing lists, inventory, etc.
Pedigree matings of selected stocks
120 to 130d
Pullets to laying cages for egg laying test
Measure body weight, sexual maturity, egg production, mortality, etc.
Revised inventory, cross identification, etc.
Initial selection
280 to 300d
Determine matings of selected males and females
240 to 280d
Accumulate egg production and other data
Egg samples for egg weight, egg shape, shell texture, shell quality, albumen quality, inclusions, etc.
Prepare initial data summaries
Estimate genetic parameters, etc.
Incorporate egg quality and other data into information system

Figure 40.7. Schematic flow chart for pure-line selection and reproduction of egg-type stock with one-year generation interval. Timings and activities vary among breeders and not all stocks conform to this pattern. Sold line = birds; broken line = information.

often recommend mass selection on the male side of broiler and turkey parent matings to achieve some further gain in growth rate, but this has often been mistaken by customers as an attempt to increase sales. There is virtually no selection applied to grandparent or parent generations of egg-type stocks.

Not only the genetic quality but also the health status of commercial stock must be monitored by the commercial breeder. The problems of maintaining freedom from disease at the primary breeders' home bases can be addressed by strict security, isolation, and repetitive monitoring using all available serological techniques. If infections manage to enter these premises, they are detected early and appropriate control measures introduced. As the generations of multiplication proceed and population sizes expand, it becomes less and less practical to impose the degree of security found at the primary breeders' premises.

Breeding stock, either parent or grandparent generation, are invariably free of *S. pullorum, M. gallisepticum, M. synoviae,* and in the case of turkeys, *M. meleagridis.* Most breeders also monitor for freedom from other *Salmonella* species. However, the ubiquitous nature of these infections inhibits breeders from guaranteeing their absence.

Maintaining freedom from these and other diseases becomes the responsibility of the customer once the birds leave the breeder's premises. Depending upon the particular circumstances, breeding stock may be maintained in the minimal disease condition for its entire life. However, there are circumstances under which this breaks down, especially in countries with a high incidence of particular diseases. Normally, breeding stock will be subjected to periodic serological tests to monitor their health status.

Various relationships exist between breeding companies and customers. In the early days of international distribution of breeding stock a system of franchising was most commonly used. Breeders appointed individual hatcheries as their independent agents for distribution. They supplied supporting advertising, promotion, and sometimes technical service. The distributor hatchery would either purchase the breeding stock outright or alternatively would make a down payment with royalties being paid to the breeder based on the number of offspring sold.

Particularly in the U.S.A. the system of using independent distributors has to some extent been replaced by breeders establishing their own hatcheries to produce commercial chicks. This, plus the emergence of large multimillion-bird integrated complexes with their own hatcheries, has led to a rapid decline in the number of commercial independent hatcheries. In many international markets breeders established subsidiary or joint venture companies in specific countries. These would operate grandparent flocks and sell parents in much the same way as the parent company did domestically. This type of distribution system applied particularly to egg stocks. With broiler stocks, the most common method of distribution is through integrated companies which control many phases of production. Here the corporation would purchase breeding stock outright and produce commercial progeny for use within its own organization. In some of the larger markets, particularly in the U.S.A., this type of arrangement also exists in the egg production industry.

NONGENETIC ACTIVITIES OF BREEDING COMPANIES

While most of the breeders' resources are directed at genetic improvement, multiplication, and distribution of stocks, significant attention is also paid to product service.

Many of the commercially important traits have low heritabilities. This is especially true of most commercial characteristics in egg layers, and those associated with reproduction in meat birds. Thus, these traits are largely determined by environment, and environmental factors could obscure small genetic differences between competing stocks. Breeders therefore attempt to design management programs which will optimize the environment, thereby permitting their stocks to perform at or close to their genetic potential.

Nongenetic factors most commonly addressed by breeders include disease control and sanitation, nutrition and feeding, and housing and environmental control especially involving ventilation and lighting. Breeders carry out a limited amount of research in these areas; this information is augmented by institutional research and, most importantly, by field experience both at the breeder level and among customers. Information relating to these factors is transmitted to customers through a variety of media, often combining marketing and sales efforts with technical service activities.

Disease control and sanitation. Disease control and sanitation play a major part in breeders' activity on their own farms. This is because it is well-known that performance differences resulting from varying disease status could be mistaken for genetic differences. Furthermore, absolute productivity can be compromised by disease incidence, thereby increasing production costs. Veterinary practices used at the primary breeder level are therefore adapted for application to commercial generations. Vaccination programs are developed and disease prevention strategies undertaken to minimize the effects of viral and bacterial infections among commercial generations.

Nutrition and feeding. Feeding and nutritional programs appropriate to each class of stock are developed by primary breeders. This presents a considerable challenge, since in the context of world-wide distribution not all customers will have the same expertise or range of feed ingredients available to satisfy the nutritional needs of commercial poultry. Nevertheless, principles are established which can be used by nutritionists in remote countries to develop custom-made feeding programs likely to meet most of the demands.

Housing and environment. Housing and environmental control are further complications in the world-wide distribution of commercial poultry. Most of the breeders are located in temperate countries and for a variety of reasons tend to use environmentally controlled buildings. These permit greater control and limit the effect of environmental variation on the ability to measure genetic differences. However, a large proportion of commercial poultry are housed in much less intensive conditions, often in tropical areas where environmentally controlled buildings are impractical. Breeders must provide guidelines to cover the entire spectrum of climatic conditions and availability of buildings.

Product specifications and guides. Breeders produce detailed specifications of all their products. The specifications include rates of growth, age at sexual maturity, anticipated rate of egg production in the case of laying stocks, egg size and quality, carcass characteristics in the case of meat stocks, and a variety of other factors. Product specifications tread a fine line between genuine information and promotional acclaim. While the specifications must represent an achievable goal at the commercial level, they cannot be seen to be inferior to those of competitors in any significant way. Thus, specifications tend to lie at the upper end of the spectrum of field data observed.

In addition to product specifications, breeders also publish management guides which describe methods of feeding, housing, etc. While these often appear to be a counsel of perfection, it is evident that if they are followed they could permit levels of performance at least equal to those specified for the products. Management guides can also be written so as to exploit major product advantages, while attempting to remedy weaknesses by means of environmental manipulation. For example, a recommendation for a stock with intermediate egg weight might be to apply lighting techniques to delay sexual maturity, thus increasing egg size. Conversely, a stock with genetically high egg weight might be accompanied by advice to induce earlier maturity, thus limiting egg size. These recommendations can be fine-tuned to suit individual market requirements and circumstances.

Management guides also contain nutritional recommendations of varying degrees of complexity. Normally, daily requirements for a range of nutrients are specified. To protect themselves from possible errors, breeders tend to recommend intakes of most nutrients towards the upper end of the range of literature values. Because the management guides are intended for world-wide distribution, they are considerably modified by those able to specify and predict nutritional regimes with a high degree of precision. Conversely, in developing countries or in locations where many feed ingredients commonly used may not be available, the management guides present a target to aim at and which may not necessarily be achieved under all circumstances.

In broiler breeders and laying hens, nutritional programs are specified along with target body weights. With the heavy meat-type breeder selected for extremely rapid growth, strict control of body weight has been shown to permit reasonable reproductive performance; anything approaching *ad libitum* feeding provokes extreme obesity, low levels of egg production, and increased mortality. These management programs have formed a major part of the broiler breeding strategy for many years. It is probably the best example of nongenetic manipulation designed to overcome the side effects of genetic progress. Severe feed restriction is necessary, and this is usually accomplished by providing strictly limited and measured amounts of feed, or by providing feed only every second day. While these techniques of restricted feeding were largely aimed at permitting continued egg production in the females, they also became extremely important for the male parents. These lines have also been selected for many generations for increased rate of growth, and even when their feed intake is restricted in the rearing period, they tended to become overweight during their reproductive life. In the mid 1980s feeding systems were designed which would exclude the males and they could then be fed a separate and specially formulated diet, while the females continued to receive their own ration designed for egg production.

Technical service. Breeders provide varying levels of hands-on technical service. Specialists visit distributors and individual customers either to foster good public relations or to provide solutions to immediate problems. This type of technical service may be performed by veterinarians or by specialists in other disciplines. The range of problems liable to confront such people is vast; experience in addition to formal training plays a large part in their success.

While technical service can be shown to serve the needs of the customer, it also serves the breeding company well. When a customer's flock suffers from a disease, there is a temptation to blame the genetics of the birds which is almost always an incorrect conclusion. Technical service can help to solve the problem as well as minimize the stigma attached to the breeder. In highly competitive industries, suboptimal environments may often be standard procedure. For example, many of the intensive egg production units provide far less than the optimum cage space known to be necessary for maximum performance. Yet producers will expect maximum biological performance from birds so managed. Breeders have to protect themselves from being held responsible for performance deficiencies which really are a consequence of a deliberate management decision and have nothing to do with the genetic potential of the birds involved. Low heritability traits like egg production are by definition highly sensitive to environmental factors and even highly heritable traits such as egg weight can be modified considerably by nutrition. Only by a continuing process of education can customers be persuaded to realize the relative importance of genetics and environment in determining ultimate performance.

BREEDERS' ACCOMPLISHMENTS

Since the mid 1950s most birds produced commercially are derived from commercial breeders. In these circumstances, it is reasonable to ask what level of genetic change has taken place. Does the commercial producer benefit from the large scale breeding programs conducted by the breeders? Rates of progress are extremely difficult to specify in the absence of any concerted effort to measure them. Commercial or field performance reflects not only genetic changes but also deliberate advances in feeding and nutrition, housing and environmental management, and disease control. Furthermore, uncontrolled factors such as disease status, seasonal and annual weather patterns, and other as yet unidentified sources of variation all contribute to observed field performance. Separating genetic improvements from this complex of causes and effects with any degree of precision is not possible.

Nevertheless, those with an intimate involvement in the poultry industry are convinced that progress has been substantial. Particularly in the broiler industry, historical trends show major gains in growth rate and feed conversion. Since growth rate is known to have a high heritability, the geneticists' claim to much of the credit for these advances seems reasonable. Two sources (Gyles, 1989; Cahaner and Siegel, 1986) illustrate these developments (Table 40.3). Based on number of days to reach a constant weight of 1800 g, Gyles (1989) indicated a reduction of about one day each year over a 38-year period. The changes in feed conversion ratio are more likely to reflect reduced body maintenance demand than real improvements in feed efficiency.

Table 40.3. Changes in selected traits in chicken broilers.

Year	Days to 1800 g	Feed conv. ratio
1950	84	3.25:1
1960	70	2.50:1
1970	59	2.20:1
1980	51	2.10:1
1988	43	1.95:1

Adapted from Gyles (1989).

Year	Days of age	Live wt. (g)	Feed conv. ratio
1935	95	1300	4.3:1
1960	67	1500	2.5:1
1986	45	1800	2.0:1

Adapted from Cahaner and Siegel (1986).

Aside from growth rate, genetic progress has also affected body conformation, providing much more attractive carcass shape, although objective evidence of this is lacking. Increasingly intensive management has placed heavy demands on the birds for disease resistance, absence of breast blisters and leg defects. Without genetic selection, it is likely that disease and condemnation losses would be much higher.

In the egg production sector, genetic progress is even more difficult to define since many of the traits under selection have low heritability and many aspects of the environment undergo substantial change over time. One possible indication of trends can be obtained from the series of random sample test reports published by U.S.D.A. from 1961 through 1977. Although most breeders changed their stock designations during this period, there were two which did not. Selected data from these two stocks and the Kentville Control, which were entered continuously in a number of tests throughout the period are shown in Table 40.4. Stock 1 exhibited nine days earlier maturity in 1975-7, while the control stock matured only five days earlier. The changes in egg production were the most dramatic, with the commercial stocks improving by 26 and 30 eggs respectively while the control gained only four. Egg weight improved 2.6 g and 1.7 g in the commercial stocks while the control decreased by 0.4 g. There was an unexplained drop in body weight of 213 g in the controls, but the commercial stocks fell by considerably greater amounts, 303 g and 372 g respectively, presumably resulting from downward selection to improve feed efficiency. The fact that in the commercial stocks egg weight increased while body weight decreased is also worthy of mention.

There is much circumstantial evidence and field data indicating genetic progress in egg-type stocks, but little or none which could withstand statistical analysis. A few breeders maintain control stocks to measure their internal rates of progress but these data remain highly confidential. To a large extent commercial industries in both meat and egg production have to rely on the fiercely competitive nature of breeding stock sales to ensure maximum genetic progress.

Developments in the field of molecular genetics in the 1980s led to speculation that these might provide the opportunity for major genetic advances in commercial stocks. However, the absence (1989) of methods to identify genes controlling commercially important traits, and of techniques for introducing new material into the genome with predictable consequences, tempered the early optimism. While commercial breeders have all

Table 40.4. Changes in selected traits in egg stocks measured in North American random sample tests from 1961-63 to 1975-77. (Data are regressed means taken from 1963 and 1977 Report of Random Sample Egg Production Tests, United States and Canada, U.S.D.A.).

	Age at 50% Production (d)		Eggs/hen housed		Egg wt. (g)		Body wt. (g)	
	1961-3	1975-7	1961-3	1975-7	1961-3	1975-7	1961-3	1975-7
Commercial Stock 1	173	164	234	260	59.4	62.0	2177	1874
Commercial Stock 2	169	164	232	252	58.9	60.6	2132	1760
Kentville Control	174	169	213	217	58.9	58.5	2132	1919

become involved to some extent in these exciting fields of research, the sale of transgenic chickens with greatly improved performance has not yet occurred. Developments in immunology, disease resistance, and vaccine production technology resulting from genetic engineering will likely precede the introduction of novel poultry genotypes.

SMALL BREEDERS

All of the foregoing sections in this chapter have, by inference, related to large breeding organizations. These companies mostly developed from modest beginnings by a process of expansion and take-over. They are almost all multinational and many are owned by large conglomerate corporations. The main exceptions to this would be the U.S.S.R. and the Peoples' Republic of China, although some of the stock in those countries is known to be descended from imports from some of the major international breeders.

There is a small but significant group of breeders who do not fit this description for one reason or another. In general, their total resources, output, and contribution to the world's commercial poultry are comparatively small, but they are significant in that they provide stock to a specific geographical region, or provide types of birds not offered by the major international breeders. A few companies are owned by governments, but mostly they are privately held. They make a small contribution to the world supply of chickens and turkeys and a proportionately large contribution to the supply of other species, such as ducks, geese, guinea fowl, pheasants and quail.

Some of the independent breeders owe their existence to political action. This may result from reluctance to use foreign currency to import parents or grandparents from the major breeders or a lack of such finance. In India, for example, importation of grandparents was prohibited for many years, while considerable public resources were devoted to development of an indigenous breeding program. Another example is Australia, where until the late 1980s a few domestic breeders supplied the entire national demand, while international breeders were excluded by a 'cordon sanitaire'.

Other breeders occupy positions of near monopoly in countries where imports are prevented due to the real or imagined risk of disease which might accompany them.

Another group of relatively smaller breeders represents commercial producers who are breeding for their own needs, because major breeders either do not produce the species involved, or are perceived to be less efficient than the small or independent breeder. This would apply to the relatively uncommon species of waterfowl and game birds. The market for these is limited, to the extent that few major breeders have considered it worthwhile to mount the necessary breeding programs.

In yet more cases, independent breeders have identified and targeted special market niches, either for less common species or particular varieties of chickens, which the major breeders have chosen not to serve. Even where the major breeders do contribute, the smaller companies may maintain business through special attention to product quality and customer loyalty.

Some of the breeding operations in countries where political actions have prevented imports from major breeders are in fact owned and operated by government agencies. This situation prevails in the U.S.S.R. and some other East European countries. Frequently, development and research conducted under these circumstances is provided by or done in cooperation with universities or other government-controlled research institutes. In this case, well-trained staff may be available to direct these programs. Conversely, in the case of the small independent breeder, there is frequently very limited professional staff and such companies often rely mainly on a few well-trained employees to conduct the breeding and selection work.

Among the small breeders much less use is made of pedigree breeding. The individuals either lack the skill and resources needed for operating a pedigree system, or they are unaware of the potential benefits such a system might confer. While the use of single male matings may be quite widespread, the need for trapnesting and egg identification creates a heavy demand for reasonably skilled labor. Subsequent record keeping, pedigree hatching, and wingbanding are further barriers which discourage those with limited resources from developing fully pedigreed populations in their breeding programs.

In these circumstances breeders who concern themselves with reproductive traits, for example egg production, face major limitations. Individual egg laying records from caged layers provide only a minimal basis for selection. If the birds are mixed half-sibs and full-sibs, some attempt at family selection is possible, but the selection of males is made much less effective than where full pedigree identification is undertaken.

In the case of meat birds, the small breeder is not under such a severe handicap as with egg production stocks. The major traits under selection have higher heritabilities and are expressed in both sexes. Furthermore, traits like body weight are easily measured, and others such as body conformation, freedom from defects, leg abnormalities, etc. are the type which expert poultrymen easily recognize. This permits such small breeders of meat birds to remain competitive and to make acceptable progress, at least for a number of generations of selection.

In most markets the commercial production of eggs, broilers, and turkeys tends to be cyclical. Periods of prosperity are quickly followed by over-zealous expansion, leading to reduced commodity prices and frequently to financial failure of some of the participants. This can be devastating for industry suppliers, including breeders and hatcheries, who may find themselves with unpaid bills, or unsaleable production, for reasons quite unconnected with genetic quality. Sensitivity to industry trends and an acute sense of timing are therefore additional requirements for the successful breeder. The large multinational breeders may have the resources to survive temporary market downturns, or be able to offset them against prosperity or new business elsewhere. The small breeder on the other hand may be unable to outlast a particular crisis.

Many of the smaller breeders failed to survive the evolutionary process. Some were taken over and incorporated into the larger more successful organizations. Others simply fell by the wayside, remembered only by the few who worked with them during their brief existence. The part played by genetics in their demise remains forever a subject for speculation.

Author's Note: Unlike most chapters in this book, this particular chapter is not a critical review of published technical literature. Such literature on applied industrial breeding and selection does not exist. Much of this chapter reflects professional experience with two international breeding companies. This included approximately ten years with Shaver Poultry Breeding Farms in Canada, and three years with Ross Poultry (now Ross Breeders) in Scotland. These provided the basis for continued fascination with all aspects of the breeding industry. Prior to embarking on this project, the author was privileged to visit a number of breeders for discussion of current trends and practices. Gratitude is expessed to Nigel Barton of Ross Breeders, Hugh Arnold and Cliff Nixey of British United Turkeys, John Powell of Cherry Valley Farms Ltd., Ad van Hedel and Theo Peters of Euribrid, J.H. van Middelkoop and D.C. Heijboer of Hypeco, Dietmar Flock of Lohmann, Jim Smith formerly of Hubbard Farms, and Bill Rishell and Verne Logan of Arbor Acres Farms for their time and patience. Informal discussions were held at various times with many other participants in the breeding industry. Their input is gratefully acknowledged. Although influenced

by all of these sources, the ideas and interpretations presented in this chapter are solely those of the author. This chapter was prepared while the author was employed as Poultry Specialist by the Ontario Egg Producers' Marketing Board. Their tolerance and forbearance in permitting this work is hereby acknowledged.

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Chapter 41. BREEDING AND SELECTION BY POULTRY FANCIERS

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The domestication of the various species of poultry began so long ago that unfortunately records are not available. No doubt early people realized the advantage of having a source of food which could be maintained largely by its own foraging together with the addition of human leftovers. Furthermore the aggressive nature of the male junglefowl could be exploited to supply entertainment through cock-fighting, while his voice signalled the break of day. Poultry were also used in sacrificial rites, often intended to increase virility.

An increasing degree of domestication was accompanied by a corresponding protection from natural predators thus offering the opportunity to influence, by accident or design, the way in which the flock developed. Mutations which in the wild were facultatively lethal, such as recessive white plumage which removed the camouflage of wild-type plumage, could be incorporated in controlled flocks.

With the first conscious human choice to influence the camp flock in a direction away from the junglefowl came the birth of the poultry fancy — the selection of stock for visual appeal. Virtually all the mutations whose effects are easily visible and which are today available for genetic analysis have been preserved through conscious selection by fanciers throughout the ages. For instance the hookless gene (*h*), whose action is to prevent the formation of hooks on the barbs of the feathers thus producing a silky-feathered appearance, has been preserved for several hundred years in the Far East and can still be found in the popular Silky breed (Hawksworth, 1982).

World exploration by sea led to the world-wide distribution of poultry as a corollary of their use as a source of fresh meat and eggs on board ship. Flocks were established near all major ports, and developed to increase their viability and attractiveness in their particular environment. Breeds of Mediterranean origin developed nonbroodiness to permit a longer laying period, because the warm climate allowed artificial incubation. Furthermore the most popular variety, the White Leghorn, has incorporated dominant white (*I*) which later was shown to be associated with higher egg production in certain stocks (Hawes, 1975).

It does not seem to be accidental that British and Northern European breeds developed with white, black, or blue legs, while those developed in North America require yellow or willow legs. The difference between them is that the latter require the carotenoid pigment xanthophyll which is absent in cereals grown in Britain and Northern Europe but present in

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Editor's Note: In past decades, poultry geneticists have largely ignored the activities and interests of poultry fanciers. That attitude is changing as an awareness of the vast genetic resources developed by fanciers is recognized. It is a resource that holds great interest and potential importance in the age of molecular genetics and genetic engineering. Dr. Carefoot, President of the Poultry Club of Great Britain, was asked to describe and interpret the activities of poultry fanciers, and the role that they have played in poultry breeding and genetics. He has become a leading fancier, and a researcher with significant contributions to the plumage color genetics literature (see Chapter 5), both as a leisure pursuit. He has endeavored to express the motives, attitudes, and opinions of fanciers, in addition to their procedures and accomplishments. Parallels with Dr. Hunton's description of industrial breeding and selection (Chapter 40) are very striking.

maize grown in the U.S.A. Could it be that while the bright yellow legs would appeal to fanciers, the difficulty of attainment without maize led to the easier alternative, a bird not requiring the presence of yellow pigment? This preference persists to this day, with the British taste being for white-skinned fowl and brown eggs as opposed to the yellow skin and white eggs fashionable in the greater part of North America.

With the advent of organized animal breeding in the 18th and 19th centuries, which led to the use of pedigrees, and perhaps more importantly to accepted breed standards, came a corresponding interest in poultry breeding with consequential attempts to create new varieties. One of the more spectacularly successful attempts was the Sebright bantam attributed to Sir John Sebright and associates in the early 19th century. Details of the blending of other varieties to produce the laced phenotype of the Sebright are most interesting and are to be found in Jeffrey (1977).

Perhaps a most important role in the development of the Sebright was played by annual meetings at which the stock produced by the breeders was assessed. The desire to have one's stock adjudged to be of higher quality than that of one's friends serves as a strong incentive to all with a competitive spirit. Such competition creates interest and determination to ensure the project's success. Fanciers with similar interests met, exchanged views, and worked to a common goal which culminated in a new variety, or breed, or a new color of an existing breed. But not all new varieties evolved in this way. Some were developed to preserve a pleasing new variation which occurred in a particular flock. Others were 'made' to satisfy the dream of their originator, and still others evolved from a desire to satisfy a particular market.

Marker genes were sometimes used as breed characteristics to prevent unwanted introduction of 'fresh blood', which, if it had occurred, may have been accompanied by a decline in usefulness to the owner of that particular flock. Famous cock-fighting lines, or in fancier terminology 'strains', were developed using marker genes as line characteristics. Cheshire Pyles using dominant white (*I*), and Lord Derby's Black-Reds which had females based on wheaten alleles at the *E* locus, are but two examples of a widespread practice. Furthermore many of the old 'cockers', as cock-fighting advocates were known, believed in breeding 'in and in' within their existing strain, always selecting for vigor for their breeding pens. They also were aware that fighting ability depended upon heredity because they made a practice of testing a hen for resistance to her intended mate, selecting only those hens which actually fought the male for a considerable period of time.

But perhaps the realization that the Sebright bantam was deliberately developed, as were most breeds, many decades before the birth of genetics in 1900, will serve to demonstrate the considerable skill, knowledge, and determination which existed and still exists in that body of people collectively known as poultry fanciers. These fanciers developed and preserved the genetic material upon which has depended much of the research into the study of the inheritance of the various species of poultry. These fanciers were not aware of or concerned with *genotypes*; they were avidly seeking particular *phenotypes*.

But who are these poultry fanciers, and what are their aims? No short definitions could answer these questions, but basically a fancier is a person who likes poultry and keeps them out of interest, not out of financial necessity. As with many hobbies, some just dabble, but in others interest increases so that poultry keeping takes up most of their spare time. These 'serious' fanciers are the ones who are not only the backbone of the fancy, comprising of skillful breeders and/or exhibitors, they are also those whose skill and success generates such a demand for their stock that from among them came the earlier 'commercial' producers and professional poultrymen, the latter often being hired by monied persons to manage a poultry farm producing exhibition poultry of economic usefulness. These fanciers developed the stock sense and practical breeding techniques which formed the basis of poultry husbandry; their powers of observation were utilized by commercial producers in the selection of their laying flocks; indeed many developed into commercial breeders and producers themselves until they became unable to compete with enormous professional concerns.

Meanwhile fanciers were producing highly homogeneous lines preserving unique alleles, so providing the basic stock with which geneticists could work. Indeed some avid fancier breeders have been so keen to improve their stock that they themselves have kept records and correctly deduced relationships between different phenotypes. Furthermore many serious fancier breeders study genetics, some with sufficient success to be able to contribute to genetic research themselves.

THE ORGANIZATION OF THE FANCY

The advent of exhibitions of livestock in the mid-19th century triggered an interest in poultry for exhibition. The exhibitors brought with them an inevitable need for an organization to enable a consistent approach, so that exhibitors and judges alike had some means of assessing their birds other than by personal prejudice. Gradually there evolved by consensus a *Standard of Perfection* for each breed to describe the ideal, coupled with a *Scale of Points* which indicated the relative importance of each point of merit in the Standard of Perfection and a list of *Serious Defects* and *Disqualifications*, examples of which are all deformities, poor feathering, and general lack of constitution. Quantitative genetic selection against such faults formed improved lines which provided the basis on which commercial producers could further improve. While there are obviously different interpretations of the Standards of Perfection, it is surprising how consistent the judging appears, and how breeds have developed variations of fashion without a need to alter the standards.

The present system of organization of the exhibition poultry fancy is remarkably similar in all countries where poultry are exhibited, and has been so for well over half a century. Invariably the following seem to be involved:

1. A national club which holds a national show, authorizes the standard of perfection of the various breeds, is responsible for publishing and maintaining a book of standards, and approves a panel of judges. The national organization also provides a detailed list of show rules and invariably is the body required to deal with alleged infringements of the generally accepted code of conduct, even to the point of being able to ban persons if they have been found guilty of serious malpractice.
2. Breed clubs which are formed by a relatively small group of fanciers who wish to foster the interest of a particular breed.
3. A rare poultry society which acts as a breed club for all the breeds and colors of breeds for which no club already exists.
4. Area agricultural societies, state fairs, etc., which hold general exhibitions of which poultry play only a small part.
5. Localized poultry clubs, fur and feather societies, small livestock societies, etc., which cater to all breeds in their own sections in local areas.

Through this common system the serious poultry exhibitors meet regularly, increase the competition, exchange both stock and ideas, and receive further incentive. Unfortunately intense competition does not always bring out the best in exhibitors, hence the need for the code of conduct and a disciplinary procedure.

By the very nature of the fancy, a collection of amateurs, while the organization remains essentially the same the personnel in office often change rapidly. Therefore while world-wide lists of breed standards books and of major societies could be produced, such lists would quickly be out-dated. Perhaps the simplest way to obtain information from another country is to contact a fancier in that country via one's own national organization.

Collectively fanciers keep all species of poultry. While much of the subject matter in this chapter is applicable to all species of poultry, discussion and examples will be confined to the domestic fowl because of space limitations.

THE ROLE OF THE FANCIER IN THE DEVELOPMENT OF VARIOUS TYPES OF BREEDS

The present era of intense pressure on commercial producers of eggs or poultry meat is far removed from the days when both economic requirements and aesthetic traits were incorporated into the breed standards. Early fanciers realized that their creations needed to be economically viable and so selection intensified the traits for meat and egg production on which the 'company' geneticist was able to capitalize.

The Indian Game or Cornish breed was developed in Cornwall, England, and attributed to Sir Walter Raleigh Gilbert who in 1846 stated to a friend that he had formed them by crossing a Red Aseel imported from India with Lord Derby's Black-Red Game wheaten females. Later Sumatra Game blood was infused to add brilliance to the plumage, and it seems that while the Indian Game was used to add depth of color to the Gold-laced Wyandotte, it is more than probable that the cross so formed was afterwards used in the Indian Game to add to its lacing (Sturges, 1921). Dominant white (*I*) was introduced into the Indian Game to replace the mahogany and black phenotype with a white and black one to produce the Jubilee color variety. Breeders of Indian Games of both colors, the original Dark and the later Jubilee, breed them freely together to this day, but frown on the practice of mating Jubilee to Jubilee because they know there will be some resultant chicks almost white. This is, of course, due to them being homozygous for the dominant white gene (*I*).

However, while color is important for exhibition breeders of Indian Game, the main criterion is the production of a flock with excellent type as per the following extract from the breed standard of perfection (Hawksworth, 1982): "Type: Body very thick and compact and very broad at the shoulders, the shoulder butts showing prominently, but the bird must not be hollow backed, the body tapering towards the tail. Back flat and broad at the shoulders, but the bird must not be flat sided. Elegance is required with substance. Breast wide, fairly deep and prominent, but well rounded. Wings short and carried closely to the body, well rounded at the points, closely tucked at ends and carried rather high in front." Any bird meeting these requirements would truly be a picture of an elegant table fowl.

An attempt to combine steady egg production with table qualities on a commercial basis resulted in the standardization in the 1930s in Great Britain of a breed with white legs (for the British market) and white feathers called the Ixworth (Hawksworth, 1982). Phenotypically the Ixworth varies only from the commercial White Cornish in having white skin and legs as opposed to the yellow of the latter. The phenotypic similarity suggests most strongly that both are descended mainly or in part from the White-laced Red or Jubilee varieties of the Indian Game (Cornish). The Ixworth however never reached commercial prominence.

As with meat producing strains, similar development took place for the strains whose main selection criterion was economic egg production. The first obvious gain was the elimination of the lost production caused by broodiness. According to Sturges (1921):

"How the non-sitting instinct arose is not known. It is probably the work of centuries. Some have attributed it to the practice of artificial incubation in Egypt carried on for centuries."

Interestingly the nonsitting breeds are usually standardized as having white earlobes, and are relatively slender in build, active, large-combed, and lay white-shelled eggs. Their standards require a length of body and a lack of coarseness which is commensurate with economic egg production. They are required to be somewhat smaller than the standard fowl not selected primarily for egg production. The Mediterranean breeds such as Ancona, Andalusian, Minorca, and Leghorn all follow the same guidelines. Indeed there is some justification for the argument that the Ancona is a mottled Leghorn, and an Andalusian is a black-laced blue Minorca, with the basic difference between the Leghorn and Minorca being the former has yellow skin and no dermal melanin while the latter has white skin and dark legs, and so they differ primarily at the white skin (*W*) and inhibitor of dermal melanin (*Id*) loci.

Dual purpose breeds. The small size of Leghorns and related breeds, with resultant poor meat yield, left a need for a dual purpose fowl which could produce a large quantity of eggs and still yield a reasonable carcass at the end of its useful life. Furthermore the fancier breeder in more extreme climates than the Mediterranean was quickly aware of the problems caused by freezing of the large combs and wattles of Leghorns. Several successful attempts were made to create such a dual purpose breed, catering to the specific needs of the area of origin. Almost all relied for their makeup upon stock of three classes, the large Asiatics such as Langshan, Cochin, and Brahma, the light laying breeds, and the more ancient localized fowl or their derivatives.

In the latter half of the 19th century in the New England states of the U.S.A., dual purpose breeds such as Barred Plymouth Rock, New Hampshire, Rhode Island Red, and Wyandotte were developed with yellow skin and laying tinted eggs. They also had precise color requirements, distinct to each breed, which enabled fanciers to make a standard of perfection which both retained their usefulness and their attractiveness. A low frequency of the recessive white gene (*c*) was present in some flocks of Barred Plymouth Rocks, which led to the appearance of white 'sports'; they were first exhibited about 1876 at the Eastern Maine Fair in Bangor, Maine by a miller named Oscar F. Frost. These White Plymouth Rocks were standardized by the American Poultry Association in 1888, and they continued to be developed as both layers of large brown eggs and as market chickens, until according to Hawes (1986): "Nearly 100% of the commercial chicken broilers being grown in the U.S.A. and in most other parts of the world have as their dam a White Plymouth Rock female. The sire side in this case is a synthetic breed, combining genetic material from the Cornish, New Hampshire and Barred Plymouth Rock varieties."

On the continent of Europe, deep brown eggs were desired which resulted in the later creation in Holland of Barnevelders and Welsummers, and in France of the Marans. The desire for dark brown eggs at first resulted in a mixed appearance but with fancier selection these breeds are now fairly uniform.

Meanwhile in England, breeds meeting the British requirements of tinted to brown eggs and white skin were also being developed. The Sussex variety was derived from infusions of imported breeds into the local table fowl, of which the Dorking with five toes was generally accepted as being the most ancient. Indeed, according to Sturges (1921), an early Roman writer on agriculture, Columella, who died about A.D. 47, described his favorite fowl and said of the hen: "Let them be of a reddish or dark plumage, and with black wings....Let the breeding hens therefore be of a choice colour, a robust body, square built, full breasted, with large heads, with upright and bright red combs....those are believed to be the best with five toes." He goes on to say: "Let the white ones be avoided, for they are both tender and less vivacious and also are not found to be prolific." It would appear that Columella knew of at least two varieties of Dorking, the Colored or Red and the White.

But perhaps the most specifically designed and recorded creation was the Orpington, named after the village in the County of Kent, England in which lived the originator, William Cook. The first variety, the Black, was specifically bred by intercrossing other breeds with the definite purpose of producing a fowl which should be black in color to enable it to withstand the smoke and dirt of suburban districts, which should be a good layer of brown eggs, and should be a useful table bird of good size, and with a white and fine skin. Sturges (1921) recorded the way this aim was achieved: "Mr. Cook says that he first mated a large Minorca cock with some Black Plymouth Rock pullets, and afterwards mated the progeny with a short-legged Langshan cock devoid of feathered shanks."

Within the space of a few short years the Orpington was spread throughout the world. Indeed such was the interest in the breed that breeders interested in exhibiting gradually altered the emphasis from utility qualities to a requirement of enormous size, and wealth of feather coupled with intense beetle-green sheen, so that the original ideal of a dual purpose utility breed was lost. So much so that around 1921 large importations of stock birds were made to Britain from Australia where Orpington breeders had chosen to emphasize its true utility type. This importation resulted in the founding of an Austral Orpington Club leading to a new standardized breed, the Australorp, which filled the vacancy left when the Orpington became an exhibition fowl, and perpetuated the ideal of the originator of the Orpington under another breed name.

The gradual alteration in the exhibition Orpington is perhaps best chronicled by Hubbard (1915). Photographs of Hubbard's Black Orpingtons demonstrated the assimilation of balance, depth, and quality of feather over the period 1908 to 1915, and his success in competition with the top breeders from Australia, Britain, Canada, and U.S.A. The determination and persistence of fanciers in their aim to achieve perfection is illustrated by the way Hubbard learned the method of producing intense beetle-green plumage sheen in both sexes by observing that both sexes of wild crows have equal sheen, thus confounding the widely held belief of fanciers that a dull bird must be mated to a bird with sheen since otherwise purple barring will result. Hubbard demonstrated that a stunting of feather growth causes the purple bar.

Hardy breeds of fowl. In addition to the creation by fanciers of breeds specifically for table requirements, or for specialists in egg production, or for the dual production of eggs and meat, fanciers have also pursued their particular interests in other ways. They have produced breeds of hardy fowl adapted to the needs of hill farms and areas of poorer land, which can partially keep themselves by foraging. In the north of England, the red-lobed Lancashire Mooneys (gold or silver with a black moon-shaped spangle at the distal end of the feather) were crossed with the less perfectly marked white-lobed Yorkshire Pheasant to produce the Spangled Hamburg. It seems these spangled fowl were coupled with the barred fowl imported as 'Dutch Everlasting Layers' under the Hamburg breed name simply because of similar breed characteristics, and the obscure reason that the imports of the Dutch Everlasting Layers came via the port of Hamburg in Germany. Less precisely marked fowl, some with interesting comb variations, appear to have been created, or rather selected, for the same purpose and probably from both common ancestors and with infusions from similar breeds. For instance the white-lobed Old English Pheasant Fowl is nothing more than an ancestor of the Gold Spangled Hamburg, the Yorkshire Pheasant. The Derbyshire

Redcap has a large rose comb as its main feature, but it is otherwise similar to the Old English Pheasant Fowl, apart from being standardized with red ear-lobes. The Buttercup, sometimes called Sicilian Buttercup, has red lobes and is otherwise a lighter colored version of the barred gold-pencilled Hamburg with a large cup-shaped comb. The white-lobed Campine is also similar to the Pencilled Hamburg but it has a wider black bar and is in possession of hen feathering genes. Further variations of the same theme are the Braekel, the Frisian, and the Fayoumi, demonstrating the many variations which were developed to satisfy a similar need, many of which used a similar phenotypic base.

Other hardy breeds were developed for more extreme climates. The Canadian Chantecler was created with a cushion comb to help it through the harsh winters. At the other end of the temperature range, in the tropics naked neck (*Na*) genes were incorporated as a breed characteristic, the reduced plumage requiring a lower protein diet but still keeping the fowl warm.

Fancy breeds. Mutations were incorporated into breeds purely because the fancier likes to preserve oddities and by so doing produces a distinctive strain, which if sufficiently appealing could generate enough popularity to evolve into a breed of its own or into a new color variety of an existing breed. As examples, silky plumage, frizzle feathering, crest, muffs and beards, feathered feet, vulture hocks, several comb variations, rumplessness, long tail feathers that do not molt, very short legs, and very long legs are variations in form which have become breed requirements. The variations in color standards are also considerable, for Jeffrey (1977) lists 79 standardized plumage colors in the U.S.A., and many of these could vary in leg color or in shade of pigment. It is therefore no wonder that great variety can be produced by specifically selected combinations of the various mutations available for the fancier to blend into the phenotypes that he desires.

The desire to produce something attractive and different was at its height in the late 19th and early 20th centuries when prize winning exhibition birds were very much sought after, regularly changing hands for sums in excess of the value of a single hen's annual output of eggs. The chances of successfully establishing a new breed were enhanced if the representatives were both attractive to the observer and economically viable in the particular area where they originated. The fanciers who by selection produced attractive economic breeds are to be applauded for laying the foundations of modern commercial flocks.

Utility assessment of pure breeds. World War I and the ensuing world-wide depression accelerated a change in assessment of the worth of poultry stock by would-be purchasers. The value of exhibition success rapidly declined, aided also by the exhibition fancier's concentration on the ideal phenotype at the expense of the breed's commercial viability. Laying trials for pure breeds were instituted and keenly fought, so that commercial breeders produced high yielding flocks which could be only recognized as following the broad outline of the breed description. They certainly would not have won exhibition prizes. Some trials assessed only production, but others were adapted to take account of feed consumption, residual carcass value, mortality, and other commercial attributes. Wins at these trials guaranteed financial success for the commercial breeders and led to the establishment of closed flocks, closed for the fear of spoiling the 'mix' by introducing fresh blood. With greater specialization and the need to keep winning at laying trials, the commercial strains became highly specialized for profitable egg production and inbred to such an extent that the crossing of two such strains produced hybrid vigor. This led to the establishment of commercial inbred lines which when crossed produced the hybrids which now dominate the egg market world-wide. Ironically, many individual records achieved by pure breeds such as Light Brahmas, Columbian and Barred Plymouth Rocks, Brown Leghorns, and White Wyandottes would rival the best of today's hybrids, but these breeds lost out because of lack of flock consistency.

In an attempt to compete, the fancy introduced 'utility exhibition' classes where great emphasis was placed on utilitarian aspects of the breed. Indeed up to the 1950s the 'cockerel classic' and baby chick show were still held as part of the Boston (Massachusetts) Poultry Show in an attempt by fanciers to keep some pressure on economic traits. Utility breeds such as Australorps, Barnevelders, Marans, and Welsummers became established as a regular feature of the show scene, as did an increased interest in the exhibiting of eggs.

At the present time in Great Britain and in Europe, the keeping of large standardbred poultry is enjoying a revival. The owners of small flocks do not find the commercial laying hybrids to be attractive. They would rather have the rarer breeds of poultry that their grandfathers used to keep. While some small poultry keepers do hatch chicks of their own, unfortunately most still wish to buy stock from breeders, which means they do nothing themselves to help preserve purebred strains. However if the wheel turns full circle, perhaps the fancier breeders of large fowl for exhibition may find they can cover their costs by supplying the needs of small poultry keepers. If so, many fanciers who breed bantams because they cannot afford the cost of breeding large fowl may be encouraged to take them up again.

FANCIER PRESERVATION OF UNIQUE ALLELES

The fancier has through the years played a significant role in providing the raw material on which the poultry industry is based. Furthermore, some fanciers also have collectively mixed business with pleasure by providing attractive fowl capable of finding their own feed under farmyard conditions. But perhaps most important of all, breeders have out of sheer pleasure conserved the large gene pool of mutations which have spontaneously occurred over the centuries. Given the freedom from international restrictions on the movement of poultry, coupled with local restrictions on keeping of fowl, there is no reason to suppose fanciers would not preserve this gene pool indefinitely. They have made a very significant contribution in that direction by forming miniatures or bantams of all but a few of the standardized fowl (Figure 41.1); those they have not are either the smaller kinds or they have never been sufficiently popular in the large version to have been considered worth bantamizing. For not all breeds which achieved standardization actually justified such an honor by being bred true to the standard to which they aspired, and consequently never really existed other than in a basic form, or in the mind's eye of their originator. It would be an error of judgement to preserve such 'breeds' at the expense of some of those that have at some stage achieved a consistent standard of quality. Similarly many colors of existing breeds have been and no doubt will continue to be standardized without ever achieving a satisfactory marriage of the new color with the existing breed characteristics. Such colors appear permanently relegated to the endangered list. But are they any loss to the cause of gene preservation if the breed is maintained to a high standard in another color, and the color is maintained to a good quality in another breed? It is not likely, but then fanciers are entitled to breed whatever they wish.

In addition to the miniatures of large fowl, true bantam breeds exist which have been selected over a long period of time for small bodies and large appendages in order to look dainty or quaint. The Sebright with its exquisite laced-tailed laced phenotype, the Rosecomb with its symmetrical head and wealth of feather, the Japanese with long tails

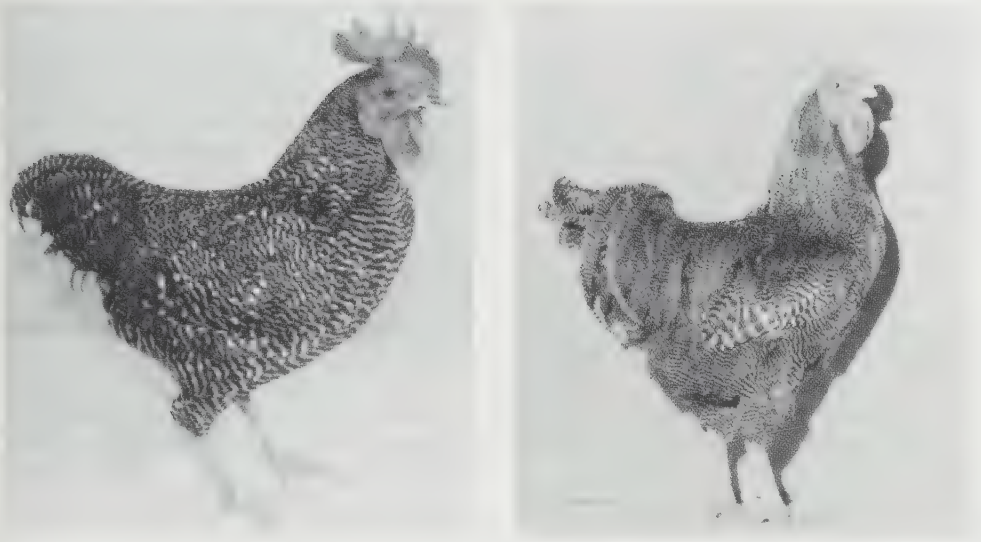

Figure 41.1. Two Barred Plymouth Rock cockerels, both heterozygous for sex-linked barring, and both show winners bred by the author. The bantam (left) demonstrates the effectiveness of the miniaturization process when compared with the standard-sized cockerel (right). (Photos courtesy Poultry World).

contrasting with short legs caused by the creeper (*Cr*) gene, the Dutch with their long wings and short backs, and the various Belgians with a multitude of intricate colors, all play their part in gene preservation and collectively present a substantial gene pool. Support for these true bantams seems never to wane due no doubt to the inherent attraction of small size coupled with the fact that they do not compete with each other for color, each basically having its own set of colors which do not overlap to any significant extent.

But perhaps most importantly of all, the fancier is preserving the genes necessary for rude good health, the genes for disease resistance. As a rule fanciers do not vaccinate. They use coccidiostats in the feed simply because they frequently cannot buy rations without them. They mingle their best stock, which are often their future breeders, almost at random with that of competitors at exhibitions. And yet it is quite rare for such stock to suffer ill effects after visiting a show, or to take illness back home. This is very different from the sterile environment needed by the commercial poultry keepers. Indeed fancier closed flocks have persistently been selected for vigor because if a bird has had a check in feathering or is slightly off form, it certainly will not win competitions or be selected for a breeding pen. A testament to the successful disease resistance of fancier-bred stock is that during the mass slaughter of commercial stock in Britain and the U.S.A. in the 1950s and 1960s for Newcastle disease, and in 1985-86 for infectious bronchitis, very few exhibition flocks were affected.

Ironically the fanciers who have been responsible for originating and developing the breeds through which such a large gene pool has been preserved are not themselves basically interested in conservation. In a review of Canadian poultry stocks, Crawford (1984) described the breeding origin of these stocks: "There are a few exceptional persons who have bred and selected a particular stock for many years, and who are known nation-wide for the quality of their birds. But most hobbyists were found to keep a number of breeds, exerting considerable selection pressure in choosing individual birds for exhibition, but not necessarily making the same careful selection of breeding stock. Instead, they may be characterized as making regular and frequent purchases of stock elsewhere." A similar observation concerning fancier or hobbyist procedures in the United States was made by Skinner (1974). Similarly in Great Britain, Carefoot (1985a) wrote: "If one studies closely each breed of poultry, then almost without exception the true breeders of any particular variety can be counted on the fingers of one hand. These dedicated few produce the birds which most of the other fanciers exhibit." He goes on to give an explanation of why this is so: "Almost without exception one can sell a very good breeding trio and within two years the new fancier is having to buy again. The reason for this is quite simple. Very few are able to build a strain for themselves because they lack the dedication and knowledge to do so, usually because of a lack of specialisation or a wish to win prizes rather than hatch chickens." The nature of the poultry fancy appears likely to be the same wherever poultry are kept.

The dedicated few who produce the strains which are generally accepted as containing most merit are continually looking for improvements in their flock. They mate breeding pens with the express desire of producing even better stock than last year. They have no wish to conserve or to preserve, but seek to change their stock in the direction they consider is needed to produce a strain of birds nearing perfection. However the most certain way of preserving a breed is by creating the demand for stock by producing quality strains. By so doing the demand for exhibition stock is substantial, ensuring that the breed is maintained.

Resynthesis and conservation. The problem of conserving a large gene pool is not easily solved. Crawford (1984) is of the opinion that "to poultry hobbyists, resynthesis is an acceptable procedure for conserving or maintaining rare breeds since their objective is to perpetuate the phenotype, not the genome which produces it." This view is well-founded and in the minds of the fancier perfectly justified simply because it was a fancier who did the original synthesis to establish the breed. So why should not another fancier repeat the original matings if he wishes to re-establish an extinct breed? However Crawford continues by saying: "The inescapable conclusion is that for purposes of the animal breeder and gene conservationist, maintenance of genetic resources contained in 'fancy' poultry breeds is not likely being satisfactorily accomplished by hobbyists, because of the apparently narrow genetic base of their stocks, and because the motives of poultry hobbyists and gene conservationists are different." Crawford's view may be correct in Canada where the usual source of exhibition birds is a very few suppliers/dealers and hence the genetic base is very small, but in larger fancier communities the genes which have been established as a constituent of a standardized breed are being preserved. Carefoot (1985a) listed 47 genes whose presence is required in at least one breed of exhibition poultry and ten whose presence is most undesirable. These were all genes with phenotypic effects and all are available in fancier-bred stock at the present time. Of course there is also an unknown number of unidentified genes, and unknown combinatory effects, but the material is there if it is desired.

In order to continue to preserve the rare breeds, a body of fanciers with similar aims formed the Rare Poultry Society in Great Britain. They instituted a scheme whereby the responsibility for the preservation of each and every rare breed was separately placed in the hands of a group of members so that the society secretary knew that each breed was catered to. In North America, a group called the Society for Preservation of Poultry Antiquities is working to preserve, with the cooperation of fanciers, as are poultry clubs throughout Europe. Unfortunately, occasionally their success is counter-productive due to a problem in satisfactorily defining rarity. In Great Britain, the Rare Poultry Society caters to all breeds which do not have a club of their own. Consequently the large fowl supported by breed clubs whose members are predominantly bantam breeders can be very few in number but not classified as rare, and therefore not the focus of attention of new entrants to the fancy who wish to keep a rare breed. The specific breed clubs are preserving these large fowl by drawing attention to their plight and usually persuading bantam fanciers to keep a few large ones. Help is also forthcoming as a spin-off from society's increased interest in antiques, causing increased interest in purebred fowl, particularly those deemed to be indigenous to the area in which the would-be-fancier resides.

As yet, societies dedicated to conservation do not appear to have established criteria for a breed or variety to be considered worth conserving, a fact which is perhaps wasting available resources. Some criteria for conservation, perhaps on distinctiveness or track record, could concentrate attention where needed and increase effectiveness. Assessment should also be made of the quality of existing specimens, and not just of their simple existence. There are many only partially established colors in some breeds, the same colors being popular and well-known in others. What is the point of 'official' conservation in such cases?

Highly homozygous strains. The fancier believes that conservation should not stand in the way of development. This opinion is especially so in that class of top breeders whom it has been established provide the remainder of the fancy with replacement stock. The exceptions are when extremely high class stock has already been produced and when the breeder wishes to conserve all the virtues and eliminate the faults in the next season's crop of young stock. In this endeavor he is successively breeding a closed flock of increasing homozygosity. Indeed when segregation at a particular locus persists it is highly likely that the standard requirement to which the breeder is aiming requires the heterozygote at that locus. The incomplete dominance of the blue color (*Bl*) locus as maintained by the standard required of the Blue Andalusian is perhaps the best known example. The observation that by seeking lemon hackles instead of the normal orange hackles in a partridge male, British Partridge Wyandotte bantam breeders invariably produced white segregants from matings *inter se* of lemon-hackled bantams, led Carefoot (1979) to show that the recessive white (*c*) gene was not completely recessive. Without the availability of such a highly homogeneous flock segregating for plumage color only at the *c* locus, it would have been extremely difficult to obtain a sufficiently sensitive background on which to test the truth of the hypothesis which many researchers suspected. Likewise the use of very specialized fancier-bred strains of patterned fowl was essential to the establishment of a single pattern gene (*Pg*), which in conjunction with various well-established eumelanin-extending or restricting genes is responsible for the plumage patterns pencilling, lacing, transverse barring, double lacing, and spangling (Carefoot, 1986, 1988). Furthermore one only needs to look at research papers on phenotypically apparent genes to realize the importance of these breeds to research, for there is a high frequency of inclusion of the remark "stock was obtained from a fancier."

It is suggested that preservation of the available highly specialized exhibition lines would benefit future research by preserving phenotypic effects which are dependent on a high degree of homozygosity. By working with such phenotypes, pleiotropic effects of known genes can be discovered. For example, the dark brown columbian eumelanin restrictor gene (*Db*) was so named because of its effect on *E* chick down. It was later shown to produce the black-tailed red phenotype in males when acting on a wild-type black-red male. Using fancier flocks, *Db* has also been shown in females to change pencilling to transverse barring, and double lacing to spangling (Carefoot, 1985b, 1988).

The availability of highly homozygous lines also permits investigation of phenotypic and nonphenotypic effects. For instance, the loci of the *Pg* pattern gene and the *Ea-P* blood group gene are known to be so close together that the two genes could possibly be allelic or even identical (Somes, 1988). Until investigated, there is no reason to presuppose the nature of their relationship, or that between other phenotypically identified genes and those isolated by study of blood groups or by other biochemical methods.

Quality — the best form of preservation. Finally, it cannot be stressed enough that the production by a skillful breeder of high quality birds of a particular variety will attract sufficient interest to ensure preservation and improvement. Failure of a fancier to produce quality stock will be more likely to deter other fanciers and relegate the breed to the endangered list. Without intending any disrespect to the fancier who wishes to preserve rare breeds which do not appear ever to have been established, when one looks at many of the rare breeds one can immediately see why they are rare. The strong and almost sentimental tendency to accept lowering of standards for rare breeds is counter-productive, for acceptance of a low standard deters fanciers from breeding them. Indeed where a fancier has taken up a breed that is in serious decline and has bred them aggressively, hatched as many chicks as possible, and rigorously culled weaklings and birds with serious defects, it has been amazing how the followers of the particular variety have multiplied. Credence must be given to the view that quality is the best form of conservation. Furthermore the current cult for preservation should not stand in the way of new developments by those breeders capable of producing something new of quality. For is not that exactly what the breeders did who originated the breeds now considered to be worthy of conservation?

BREEDING — A FANCIER'S APPROACH TO GENETIC IMPROVEMENT

The fanciers who originated many of the most popular breeds, and indeed those who have produced outstanding strains, combined keen observation with an inbuilt belief that all traits were inherited unless proven otherwise. They knew that many of their mistakes were caused by 'factors' inherent in their strains. This knowledge led to a healthy caution in selection of their breeding pens. They certainly were not students of poultry genetics, and even if they had been they would likely not have benefited from the knowledge which was available in their day. For once a breed has the required established genes fixed within it, the accumulation of minor genes (or modifiers) is what exhibition poultry breeding is about. And all that a study of genetics would teach the breeder is what he already knew — to assume all things are inherited until proven otherwise. Collectively, through sharing common knowledge in interested and often heated discussion at exhibitions, the keen breeders learned the practical rules of their trade.

The observational powers of the successful fancier breeders are therefore necessarily quite highly developed, and extend to other aspects of poultry breeding not immediately obvious. For instance, many fanciers have a limited budget available, for the simple reason that they are self-financing. Many also wish to exhibit their best stock at peak fitness in a particular show, usually the annual breed club show. Consequently they have to time their developing stock to perfection so that their exhibits are just at their peak; this means knowing when to hatch and therefore when to get their breeding pens to their best breeding potential. Hubbard (1915) reported that he had experimented a great deal with the best breeding age of a female in his strain of Black Orpingtons, and concluded a hen or pullet of ten or 11 months will breed her best chickens after she has been allowed to set (go broody) for three weeks, been broken off, and put back into the breeding pen. The first 5-6 eggs she lays after putting her back in the breeding pen will be a little small in size and will produce only fair chickens. As soon as her eggs are uniform in size, they will produce the best chicks of the season until she shows signs of becoming broody; from then on the chicks hatched will be the poorest lot of the year. Hubbard's record of wins from rearing only 200 chickens a year, in highly competitive days, lends credence to his observations.

The most important ability possessed by successful breeders is the ability to 'see' the quality of the bird at which they are looking. The fancier seeks to produce attractive birds, with the definition of 'attractive' depending on the breeder, unless of course one is trying to perfect an existing breed when the accepted interpretation of the standard of perfection is the aim. In theory the standard of perfection should give the picture of the bird required. Unfortunately this is not the case, for the simple reason that these standards are so concise that they do not permit a sufficiently detailed description of all the attributes of a true champion.

An underlying essential quality of a top-class fowl is balance. Without balance no bird can be really appealing, for it cannot help but appear ungainly or awkward in some way. The further out of balance the bird, the more awkward looking it appears. For this reason it is essential to assess poultry on the ground. It must be appreciated that in any chosen bird, balance is there, or it is not. However in order to breed balanced birds from birds not so blessed, the breeder has to be able to assess the reason for the imbalance. The reason is quite simple — the skeleton is the wrong shape, but how or where it is incorrect is not quite so easy to ascertain. The most important single reason for imbalance is incorrect positioning of the shoulders, causing undesirable alteration in the carriage of the wing, which then alters the relative distribution of weight. To be balanced the length of shank and thigh should be equal. It is rare that the length of thigh exceeds that of the shank; the imbalance is almost always the other way around. Elongated shanks lead to stiltiness because the fowl pivots about its hock when walking, with the result that a stiffness develops to counterbalance the incorrect distribution of weight over the feet.

Imbalances are mainly the result of faults observed in the extremities, in addition to altered stance of the bird. However it is possible for a bird to be faultless in back, legs, width, and shoulder position, and still be out of balance. The lengthening or shortening of any and every bone alters the weight distribution and therefore the balance. Consequently it is not surprising that a frequent comment about winning birds is that they 'read well'. By this is meant that all parts are in proportion to each other.

For perfection to be achieved, perfect feathers must grow from the perfect body. Almost all breed standards are at best incomplete in regard to their description of feather, and some give the impression that the compilers have avoided the issue altogether. The feathering ought to compliment the body in forming the shape of the bird and the desired outline. Unfortunately some fanciers tend to compensate for a narrow shallow body by adding overly profuse feathering, so that the overall bulk is approximately correct. This approach is doomed to failure because the increase in bulk of the feather also adds bulk in unwanted areas.

The precise feather shape which the breeder seeks to attain will depend on the type of markings which the chosen bird ought to possess. For example, in the United Kingdom, the round lacing standardized for Silver and Gold-laced Wyandottes requires a round end to the feather, while the almond-shaped lacing of the Sebright requires a correspondingly shaped feather. A Barred Plymouth Rock is required to have transverse bars ending with a black tip. If the end of the feather were round, a semicircular tip would result, conflicting with the bars in the feather proper. Consequently in many of the best British Barred Plymouth Rock females, a flat end to the feather has evolved in order to exhibit the barring more clearly.

To complete the picture, account must be taken of the color needed to be produced in the feather. Uniformity of color and markings is essential if an impression of quality is to be created, since in supposedly uniform breeds any obvious variation destroys the overall effect. For uniformity to occur it is essential that the development of the bird has been unchecked, because alteration in feather growth rate can have far-reaching effects. In breeds which produce both black and red pigment, a severe cold can cause a red line to be produced across the black core of the hackles. The pattern can also be changed by an alteration in the growth rate; a check can cause a pencilled feather (concentric rings of black entirely contained within the feather) to be transversely barred, and can cause a laced feather to develop the internal black pigmentation which fanciers know as 'mossiness'.

In order to breed a bird of true quality it is necessary to form a blend of birds which are inferior in some respect. The finding of the correct blend is the true secret of breeding, but the search for such a blend is none too easy. However a vision of the desired end product, with a knowledge of the faults in the existing stock, coupled with the ability to predict the possible outcome of matings, and incorporating disease resistance so that growing stock may attain its full potential, is a sound basis with which to start. Determination to succeed by persisting with the best stock available, and hatching plenty of youngsters, will steadily bring the goal nearer.

Infusion of new genetic material. The collective achievements of fanciers in originating, developing, and conserving the breeds and highly specialized lines discussed above did not happen by accident. They occurred as a result of the basic interest and determination to succeed which all achievers must have, coupled with the ability to relate their observations of the stock into an accurate assessment of its breeding potential. Since fancier breeders are trying to produce something which does not already exist, they are prepared to try adventurous crossbreeding, both in order to produce a new breed, as seen in the blending of the Langshan, Minorca, and Plymouth Rock into the Orpington, and to improve an existing breed. In the second case the breeder also maintains the stock in its original form until breeding tests prove the new infusion has been successful. An example of the introduction of a Black Wyandotte bantam into Barred Plymouth Rock bantams with the express purpose of improving the type of the Rocks was reported by Carefoot (1985a). The introduction was so successful that the Barred Rock bantam pullet which was awarded Best Barred Rock bantam at the 1978 British club show was a great-great-granddaughter of the Black Wyandotte. This winning pullet was also dam of the winning pullet at the 1979 club show, and granddam of the winning pullet at the 1980 club show, thus confirming the success of the infusion of 'fresh blood'.

Single sex selection. Frequently a high degree of competition was accompanied by an intense concentration on the production of the desired bird. If the required characteristics were only manifest in the males of the flock, then a careful study would be conducted of the females which produced the males closest to the ideal. As the males became closer to that which was desired, their mates became more uniform. In other words, these developed females had assimilated the genes necessary to produce the desired phenotype in the males, and by so doing had developed an amended phenotype of their own. These distinctive specialized females whose sole purpose was to produce cockerels of desired phenotype were called *cockerel-breeding females*. Conversely, if the required phenotype was to be found in the females, a parallel situation would result in cockerels whose main purpose was to maintain a female line with the desired quality, and hence would be *pullet-breeding males*. As pointed out by Carefoot (1985a), a popular misconception is that the description cockerel-breeder or pullet-breeder implies that there will be a large proportion of chickens of the sex referred to in the description. Obviously this cannot be so, otherwise commercial producers would have developed strains in which 95 percent of the chicks were females, thus saving enormously on the cost of producing a female chick from an egg laying strain.

The terms pullet-breeders and cockerel-breeders are most commonly used in the exhibition section of the poultry community, simply because both lines occur within the same breed descriptions, the terms being used to distinguish between them. But the practice of breeding for the properties manifest in one sex only are practiced more frequently in other sections without specific reference to the fact. Cock-fighting strains are cockerel-breeders, as are the long-crowers developed in various parts of the world. According to Wood-Gush (1959), an example of continued selection for crowing is to be found in Turkey where some males are capable of crowing continuously for periods of up to 30 seconds. The demand by anglers for feathers with which they can produce lures to imitate naturally occurring flies has also led to strains of cockerel-breeders specifically bred for their capes. These capes are required to consist of feathers of desired shape, length, stiffness, and color, and consequently their production depends on females which can breed males with high-grade capes.

On the other hand both fanciers and commercial breeders, who make conditions on the quantity and type of eggs they wish their flock to produce, are selecting for the ability of their females to meet the desired requirements. The males are only required to pass on or enhance the egg producing ability of the females and as such are pullet-breeders. Similarly, strains of domestic fowl have been developed by pheasant and other gamebird rearers purely and simply for their ability to hatch and brood chicks. Such strains are also pullet-breeders since not many cockerels go broody.

Cockerel-breeding and pullet-breeding lines are essential in some breeds of exhibition poultry simply because the desired standard of perfection described for each sex is incompatible and does not make genetic sense. The most glaring examples of the mismatched standards are in marked varieties such as the Partridge Wyandotte, where the cockerel-breeder is a black-red and pullet-breeders are gold-pencilled. Detailed descriptions of the cockerel-breeding female and the pullet-breeding male are to be found in Carefoot (1985a). Pullet-breeding lines of Pencilled and Spangled Hamburgs have even been developed with hen-feathered pullet-breeding males to enable assessment of the quality of markings on the male, which would not be so clear on the normally feathered male. Ironically, the practice of having two lines, a male line and a female one, is called 'double mating', even though it is the supreme example of single sex selection.

Sex-linked barring as found in the Barred Plymouth Rock has a dosage effect whereby the homozygous male has a white bar twice the width of that in the hemizygous female. Thus the standard requirement of equal bar widths of black and white has led to the fancier producing two different solutions to the problem.

The first solution is to have two lines. A cockerel-breeding line is selected for darkness so that the homozygous male is as dark as possible in order to approach equal bar width, while his mate approximates a 2:1 ratio of black to white. A pullet-breeding line is selected so that the hemizygous female has bars of equal width, while her mates have a 1:2 ratio of black to white. Indeed these lines are standardized in Australia and New Zealand as two breeds, Darks and Lights. This method of attaining equal bar width ratio is widespread. However, in continental Europe the light phenotype appears to be accepted in practice and in preference to double mating.

The second method of attaining equal bar width on both sexes was adopted in Britain soon after importation of Barred Rocks from the U.S.A. in 1872. The heterozygous male is the exhibition bird (Figure 41.1) and matches his hemizygous mate, both having approximately equal bar widths. However the drawback to this method is that they do not breed true. The mating of an exhibition pair produces sports in the form of light (homozygous) males and black females. These sports can be successfully used in breeding programs (Carefoot, 1985a), in much the same way as blacks and splashed birds can be used in the breeding of Blue Andalusians.

These examples of wide natural variations between the sexes serve to illustrate what is to the exhibition breeder a far more serious obstacle to achieving high-class stock than may be apparent to the casual observer. The 'natural sexual differences' often referred to in the description of perfection almost invariably prevent the ideal of both sexes being compatible. Thus some slight double mating is practiced in almost every breed, including those purported to be able to produce winners of both sexes from the same pair.

Fancier strain-making. Assume now that the breeder/fancier has carried out an accurate assessment of the available breeding stock. The breeding pens have then to be selected so that there is the maximum chance of high-class stock being produced. The expert breeder knows that many birds will be produced with a collection of varying numbers of unacceptable faults if any major improvement is to be made in the best offspring, for if desirable genes are to be assembled together on the same bird, other birds will almost certainly accumulate faults. In order to achieve improvements, the breeder accepts the fundamental principle in breeding that one should obtain the *maximum number of chickens from the minimum number of parents*, those selected for quality.

The best available breeding stock is not necessarily the best all-round exhibition stock. Frequently regular winners are birds which are not 'too bad' anywhere and beat the possessors of exceptional qualities accompanied by serious faults. However, provided mates can be found to enhance these exceptional virtues without reproducing the serious faults, the latter bird is far more valuable as a breeder. Consequently a bird's success in the show pen is not an assurance of success in the breeding pen. A balanced pen is the aim, not the mating of big winners. In the making of a strain intended to produce a regular supply of high quality stock, breeders may even have to embark upon a series of matings intended to produce future breeding stock, so that they can have the stock available to achieve the ultimate aim. One must not lose sight of the fact that all the virtues required to be assembled together to make a perfect bird must be retained somewhere within the strain.

The breeder is well aware that once a particular virtue is 'fixed' within his strain, then provided all birds in a given pen are in possession of this virtue, a high proportion if not all of the offspring will also possess this desired characteristic. Such regular repetition of noticeable points has led to the principle that *like begets like*. The breeder knows that unfortunately faults are also inherited in exactly the same way, so that the mating together of birds with the same fault will perpetuate the undesirable. The skillful breeder should have developed a strain where all the 'fixed' characteristics are desirable, and the variation occurring within the strain allows the future blending of all desirable qualities onto a single bird. In other words, it is possible to produce perfection.

As the breeder knows to his cost, two birds both of which possess the same desirable characteristic may produce offspring in which a proportion do not possess the same virtue. These are called 'sports' by the fancier. The production of sports is an example to the breeder that like does not always breed like, and that breeding is much too complex to be adequately summarized in three words. The breeder must have a knowledge of the faults and virtues carried within the strain so that an assessment of the likely outcome of a certain mating can be predicted. Successful breeders frequently 'try' a male with a few females before the breeding season in order to assess his likely breeding potential. This testing procedure, or *progeny testing*, is well-known to the poultry fancy but it has very little direct use, being confined only to the elimination of carriers of specific undesired characteristics. It does, however, serve as a useful reminder that the visual attributes of the prospective breeding stock are sometimes misleading, even though the assessment of such visual attributes is frequently the only practical way the fancier can select his breeding pens. However the fancier automatically practices indirect progeny testing when individually selecting the stock for the breeding pens the following year, for if the progeny from one pen is better than that of another, the former will have more representatives in future breeding programs. After several years of selection any bird which produces unsatisfactory offspring will automatically have such progeny discarded and therefore its influence on future generations is largely removed.

The exhibition poultry breeder places a considerable importance on knowledge of the ancestors of a proposed breeder because the bird under consideration may exhibit faults but carry virtues; that is, it may possess a single dosage of desired recessive genes. For this reason a poor bird from a good strain can often breed better than an odd good bird produced from a family of mediocrity. Consequently the fancier knows any selection of breeding stock can only be based on knowledge of likely breeding potential, this knowledge being gleaned from the characteristics of its known ancestors coupled with the guide given by visual observation and experience of the birds in question. For this reason the breeder of high quality stock knows the mating of related stock, or inbreeding, is essential if a strain producing stock of a consistently high standard is to be produced and maintained, and further, that inbreeding is the surest way of fixing both good and bad factors in the strain. Hence one must select so that the good is assimilated while the bad is discarded. Desirable points within any strain include health, vigor, longevity, disease resistance, ease of reproduction, and temperament, in addition to phenotypic requirements.

Every breeder is well aware that this aim is easier to achieve in theory than in practice, one of the major problems being that one cannot tell without breeding tests whether selected breeders carry the tendency to develop specific weaknesses. Thus inbreeding will produce its share of weaklings, a fact which has led the uninitiated to believe that inbreeding is to be avoided. Judicious inbreeding therefore requires that within the strain plenty of chicks are hatched, with all unthrifty stock being culled, for the breeding from stock of doubtful vigor is highly likely to create enormous problems within a few years.

The breeders of the most outstanding exhibition strains largely remove their concern about the possible detrimental effects of inbreeding by extremely careful selection. They are well aware of the need to select against any and every fault which occurs within the strain, and practice the *technique of minimum attainment* described by Carefoot (1985a). This principle is to set a quality standard for each required point of excellence. When the breeder is able to raise the minimum level for a specific point without decreasing the acceptable level for others, the strain has been improved. By the use of this piecemeal approach to excellence, considerable improvement can be made without the risk of related inherited faults also becoming fixed within the strain. Prevention of faults is the breeder's aim if one is seeking perfection and a faultless bird.

There can be no hard and fast rule with regard to the degree of inbreeding which can be practiced before inherited weakness causes serious problems for the strain-maker, for the unknown genetic background with regard to these weaknesses is the key to the problem.

However the body of fanciers has learned by experience to accept and adopt several empirical rules which collectively have stood them in good stead. Firstly, they select for vigor and prefer each year to use a cockerel with about four yearling hens. In this way they have selected their breeding females in their pullet year for phenotypic qualities, while after the pullets have proved their vigor and disease resistance, and demonstrated their laying ability, a second selection for phenotypic qualities establishes the most likely breeders of high quality vital stock. By mating them to a cockerel, chosen to blend with the hens while homozygosity for phenotypically apparent genes is preserved, almost random distribution for other genes can be maintained. For the cockerel was not bred from his mates; his dam was one of the yearling hens used the year before, and therefore since they are yearlings they are not the dam of any of his mates. If this procedure is repeated annually, then after several years the degree of inbreeding can be quite small, and can be decreased still further by an increasingly popular practice of fanciers where they have two cockerels, suitable for the hens and with little to choose between them, that are placed with the hens on alternate days. This practice has the added advantage of increasing viability because the most fertile cockerel will be more likely to have a larger number of offspring, while the one breeding the most vigorous stock will be likely to have more offspring available for selection for future breeding pens.

Fanciers often include an older hen of outstanding merit in their breeding program simply because they feel she is too good to waste. While this may increase the degree of inbreeding, if this results in a reduction of vigor then by selection the weaker offspring will be eliminated. But if the resultant retained stock is of higher quality, her inclusion is justified.

Of course there is no reason why a particular male should not be used for several seasons, provided he is not mated with successive generations of his daughters. In fact if he is kept with the same hens which were used the first year he bred successfully, there is no problem whatsoever. Any male which deserves the honor of repeated selection for the breeding pen will presumably have been considered of high quality, and in addition ought to have produced an unusually high proportion of good quality offspring, for if he had sired moderate stock he would surely have been discarded.

The fancier recognizes the relationship between the ability to produce an unusual degree of resemblance in one's offspring (*prepotency*) and the homozygosity of a very high proportion of genes producing the desired virtues or preventing faults. The term prepotency is usually reserved for males simply because the mechanics of reproduction allow a far greater number of offspring to be sired by a male than can be produced by a female. Prepotent males consequently command a healthy respect and feature strongly in many animal breeding programs, while females are frequently only considered as a means to an end, the production of the next prepotent males. However, in exhibition poultry breeding, pens are often especially mated to produce high quality stock of one sex only. When that sex is male, the aim is to produce a sequence of prepotent males, and when it is female the purpose of mating is to produce a number of high-class females each producing a large proportion of pullets of even higher quality, that is, a sequence of prepotent females.

Because prepotency is associated with a minimum number of unwanted recessive genes, the greater the homozygosity of the stock for desirable genes, the greater is the likelihood of prepotent birds being produced. The production of a sequence of prepotent breeders is therefore both a sufficient and a necessary condition for the establishment and maintenance of a strain producing a regular supply of high-class stock. For this reason many excellent breeders breed from their best cockerels and prefer to molt them and either exhibit them or keep them in reserve in their later years, even if they have bred outstanding stock. For if they were worth retaining for breeding a second year, they must necessarily have themselves produced high-quality cockerels, in which case these cockerels should be used instead of their sire.

From time to time an outstanding bird is produced, one so outstanding that a breeder may decide to strive to obtain as close a relationship to this superb specimen as possible, or as the fancier would say, to preserve a high proportion of the 'blood' of this superstar. This aim can be achieved without extreme inbreeding by the mating of more distant relatives, all of which must carry a high proportion of descendancy from the exalted individual. Charts and diagrams have frequently been published (see for example Hubbard, 1915) demonstrating methods of achieving the desired proportions of descendancy (or 'blood') from a certain individual. This method of attempting to produce high-class individuals is called *line-breeding*. Unfortunately the lifespan of domestic fowl is often insufficient for stock of the desired relationships to be available, and consequently the schematic plans fall down.

Any attempt at practicing the necessary discipline for obeying the prescribed relationships required by any line-breeding system causes the breeder to choose between relationship and quality. The master breeders are well aware that quality must always come before relationship, and therefore the breeder must over-ride the master plan whenever all the birds of a particular relationship to the superstar may be inferior to other available stock. The degree of line-breeding is therefore reduced but the quality of the progeny may be enhanced. So to some extent line-breeding places undue emphasis on former outstanding birds, and at the same time it restricts the selection process among current stock. Further, it makes an assumption that the degree of relationship to the former superstar is most important, while in practice the breeder wants to preserve the virtues of that outstanding specimen within his current flock. Because of the discontinuities in inheritance, visual likeness does not accurately reflect the degree of relationship between two birds.

In practice line-breeding in a modified form is useful to the breeder seeking top-class stock. Line-breeding by obtaining a large number of chicks from the superstar, and then mating these offspring in such a way as to permit the maximum chance of the required genes recombining in the desired quantities to produce further outstanding specimens, is wisely practiced by those breeders fortunate enough to have a breakthrough in their search for perfection. But then, if necessary, line-breeding to the best of those new outstanding specimens is to be advised rather than trying to preserve relationships to the first superstar. Of course line-breeding is rendered obsolete as a method of breeding once the strain has achieved the aim of the breeder — a flock of consistently high standard. For how does one choose a superstar in that case?

Application of breeding principles. The practical approach to the use of breeding principles as applied to small closed flocks is to be aware of them all and to use them to the best advantage that circumstances will allow. The successful strain-maker knows that one must:

1. Hatch plenty of chicks from the mated pens.
2. Select at all times for vigor, temperament, and other important traits.
3. Select the highest quality birds for breeding, being careful not to breed in faults.
4. Line-breed to the extent of obtaining a considerable number of chicks from an outstanding bird, mating these so as to preserve both a high degree of quality and a high degree of relationship to the original superstar in the second generation, but then select the outstanding offspring and line-breed to these, not to the original.
5. Recognize the relationship between prepotency and homozygosity for desired genes, and therefore consider the disqualification as future breeders of the stock which contains

only a small proportion of good birds, since any good bird whose sibs are poor is highly unlikely to be prepotent; that is, progeny test.

6. Endeavor to ensure that in the selection processes, all desired virtues are evident somewhere in the strain so that they can in the future be blended into the perfect bird, remembering the 'like begets like' principle.

Together with the ability to correctly assess the quality of stock, the application of these six points is the key to successful breeding. The knowledge of which point to apply in a particular circumstance comes from experience. In fact top-class breeders automatically assess their stock for breeding potential by combining visual pointers with known ancestry to predict the likely outcome of a particular mating. Such breeders are attempting to assess the genes carried unseen within the flock so as to control the results.

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Chapter 42. BREEDING AND SELECTION PROGRAMS IN DEVELOPING COUNTRIES

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The developing countries of the Third World are characterized by a wide diversity of climates, topography, population densities, cultures, and farming systems. Differences are also pronounced with regard to the stage of poultry development among these countries. According to U.S.D.A. statistics (1987), Brazil and Thailand are emerging as major poultry exporting nations of the world. But the poultry industry in most of the developing countries is still very underdeveloped due to lack of capital investment and feed resources, improper health control, and weak bargaining power in buying and selling. F.A.O. statistics (1985) reveal the poor production performance of three major kinds of poultry in developing countries compared to developed countries (Table 42.1). This disparity is more glaring when one considers the Third World as a place in which 75 percent of the planet's population lives, where 86 percent of all children are born, and where 98 percent of all child and infant deaths occur (W.H.O. statistics, 1985).

Local domestic fowl and ducks in developing countries still contribute to a high degree towards meat and egg supply, in spite of the distribution of high yielding stock from developed countries to these regions (Horst, 1988). An African report (Akinwumi, 1979) showed that 92 percent of poultry products in Nigeria are derived from indigenous poultry stock, and an Asian survey (Prawirokusumo, 1988) stated that about 40 percent of the egg production and 30 percent of meat production in Indonesia is contributed by the local type of chicken. In India, 70-75 percent of the poultry population still consists of local types, although they only provide 18-20 percent of the total egg production in the country. Duck populations of India, Bangladesh, Indonesia, Malaysia, and Thailand are mostly indigenous types, although international commercial breeders have recently been marketing their products in southeast Asian countries.

Horst (1988) claimed that products from local poultry stocks are widely preferred because of pigmentation, taste, leanness, and their availability for special dishes. He argued that despite the important role of local poultry, there is very little information on its genetic makeup with respect to performance, its comparative evaluation with imported lines under similar management conditions, and its adaptability and resistance to local diseases. Therefore, the potential significance of local poultry for future breeding strategy, and as a supplier of major gene complexes or single genes affecting special traits or additive and

Table 42.1. Poultry census (in millions) in developed and developing countries. (Modified from F.A.O., 1985).

	Developed countries			Developing countries		
Year	Chickens	Ducks	Turkeys	Chickens	Ducks	Turkeys
1983	3838	35	182	3976	131	25
1984	3858	35	183	4178	132	26
1985	3924	35	190	4346	135	26

nonadditive genetic variance, is still not known.

TYPE AND PERFORMANCE OF INDIGENOUS POULTRY

Chickens. Indigenous domestic fowl in many developing countries possess a similar appearance, but within local populations a great variability in morphological characteristics always exists (Horst, 1988). Comparative studies of strains from different regions within a country, for instance in Nigeria (Nwosu, 1979) or Ethiopia (Forssido, 1986, as cited by Horst, 1988) demonstrate similarity in body weight and egg performance. Estimates of genetic distances involving loci for plumage color, blood groups, and blood proteins among strains of Deshi chickens of Bangladesh were low, suggesting that the native chicken of that country may be regarded as one population. Genetic distance between some native breeds of other Asian countries is also comparatively low (Okada, 1987).

Local chickens are mainly used as dual purpose birds, although there are breeds specialized for either meat or egg production, for instance Assel in India for meat, and Egyptian Fayoumi and Deshi of Bangladesh for egg production (Horst, 1988). Body types of indigenous chickens are classified as dwarf, normal, and heavy (fighters) whose adult body weights are 800, 1400, and 2000 g respectively (Ghat et al., 1980; Horst, 1988). Most of these nondescript unimproved chickens are characterized by small body size, late maturity (up to 250 days), low yield in egg number (20-150) and egg weight (25-55 g), small clutch size (2-3 eggs), long laying pauses, and an instinctive inclination to broodiness after about eight eggs are laid. Fertility and hatchability for the eggs of these native stocks usually are much higher than in exotic birds. Egg quality characteristics are favorable in respect to breaking strength (Valle et al., 1988), yolk percentage, and cholesterol content within distinct local strains (Horst, 1988).

Shell pigmentation is predominantly tinted or brown (Horst, 1988). Plumage pigmentation tends mainly towards black and brown colors, showing extended and pied colorations. Plumage distribution is mainly normal, with special forms such as crest, ptilopody, and naked neck appearing sporadically. Exceptions to the generally observed normal feather structure are frizzling and silky feathering. Shanks and skin are frequently pigmented, showing black, green, and blue variants. Additional melanin deposition in skin, muscles, internal organs, and bones can be found not only in the silky fowl but also in other Chinese and Taiwanese, Thai (Samae Dum), Indian (Kadakanath), and even in Caribbean local strains. Dark meat chicken is regionally highly valued because of its supposed dietetic value and medicinal properties; it is assumed to cure bone and kidney diseases, chloromic disease, and impotence. Single comb prevails, but rose, pea, walnut and duplex combs occur sporadically. A long list of morphological marker genes can be found in native domestic fowl. Experimental work on some of these genes for their utility in a tropical climate has been undertaken by Horst (1988).

Adaptability of local chickens to unfavorable environmental conditions is usually stated to be high, although the underlying mechanism is not yet clarified. However, resistance to prevailing diseases such as Newcastle disease, fowl pox, and coccidiosis is low, resulting in generally high juvenile and occasionally high adult mortality rates under extensive husbandry systems.

Ducks. Nearly 72 percent of the world's duck population is found to be under extensive and semi-intensive production systems in south and east Asia (Hetzel, 1982). Indonesia, Taiwan, Thailand, India and Bangladesh with a duck population over 16, 10, 19, 9 and 6 millions, respectively, are large producers of dual purpose ducks (meat and eggs). In Indonesia, Bangladesh, and eastern India, 30 percent of eggs consumed are duck eggs. Distinction is seldom made in census records between domestic and muscovy ducks and their hybrids.

In contrast to chickens, very little work has been done to study the presence and effect of major genes on characters related to production performance in other species of poultry. However, tremendous variation exists in plumage color of Alabio and Tegal ducks in Indonesia, Itek Java in Malaysia, Deshi in India and Bangladesh, and En Si Ma Ya and Miang Yang Ya in China (Mukherjee et al., 1986). A thorough study of plumage color genotypes and their interactions is necessary before planning a breeding program involving the native ducks and their exotic contemporaries.

Geese. There are many local goose breeds in China and other parts of Asia. According to Yang and Wu (1988), China has 12 goose breeds which include Yangjiang, Quingyang, Taihu, Xiupu, and Shitou. The average annual egg production of Taihu is 60 eggs, with an average weight of 133.8 g. One of the heaviest breeds of geese in the world is Shitou, with adult male and female weights of 10-12 kg and 8-9 kg, respectively. Zie, a local goose strain of Heilongjiang province of China, produces about 100 eggs annually, which probably makes it the highest egg producer in the world (Yang, 1988). The Huoyan goose, another breed famous for its egg production (80-120 per year), originated from Changtu in China (Ning and Qu, 1988).

Japanese quail. There has been increasing activity in the production of quail eggs in the developing countries of Asia, because quail breeding and production offer an opportunity for diversification, an easy management system, and an early marketing age. Much of the parent stock has been imported from Japan. They are kept as a dual purpose bird for eggs and meat. Most farmers, however, concentrate on egg production. A bibliography on Japanese quail research work done at the Central Asian Research Institute, Izatnagar, India has been compiled by Srivastava (1987).

Other poultry species. Although guinea fowl are an important source of eggs and meat in Africa, there is very little information available on their numbers and distribution. They are distributed in very small numbers in various parts of southeast Asia. Turkeys have only a small role in poultry production of developing countries, and ring-necked pheasants are almost totally unknown.

BREEDING PROGRAMS

There are two parallel chicken industries in most developing countries. There is a tremendous potential for improving and increasing local poultry through small-holder schemes, the success of which depends essentially on improvement of genetic and nongenetic components of the industry. Conversely, some people argue that in those developing countries which have the infrastructure for massive growth of the chicken and duck industry (for instance far eastern and southeastern Asian countries, the Middle East, Brazil, India, Pakistan), a faster rate of genetic progress could be made through multiplication of imported stock and their distribution to farms having intensive, semi-intensive or even extensive (small-holder) systems of management.

There are many arguments against the latter suggestion. Barker (1982) argued that there are large phenotypic and possibly genetic variations existing within the indigenous breeds and varieties, and therefore the application of genetics toward improving these stocks should be undertaken through proper evaluation and documentation of these breeds, and a suitable selection procedure has to be devised to provide an optimum genotype to the farmers. Most of the conservationists (Arboleda, 1987) support this proposal because rich and well conserved natural resources give a country great opportunity to sustain economic growth and development, and give its people a strong feeling of self reliance, sense of pride, and identity. Furthermore, many experiments conducted by researchers in developing countries (for instance Mukherjee and Chai, 1987) revealed differential response of genotypes in different management systems for body growth, egg production, and carcass characteristics. Their results indicate that specific genotypes should be selected for optimum production within particular management and feeding systems.

Most of the developing countries recognize the need for alternative breeding strategies for poultry improvement. These strategies are listed here, with elaboration to follow.

1. Development of village chickens and ducks through purebreeding and selection.
2. Gradual replacement of native males by locally adapted and genetically improved males derived from exotic stock.
3. Use of local genomes and major genes in tropical synthetic line breeding.
4. Production of broiler and egg laying progeny from a two-way cross of lines (parent stock) at the elite nucleus flock level (regional centres). In most of the advanced nations of the Third World, this role is undertaken by parent stock breeders in the private sector.
5. Production of broiler and egg laying progeny from a four-way cross of grandparent lines. Grandparent breeders in different countries (specialized elite nucleus flocks of government or private breeders) function with the possession of four lines, either imported from overseas or as synthetics derived locally, as in the case of South Korea, India, Malaysia, and Thailand.
6. Production of pure lines from which grandparent stocks and parent stocks are derived. Countries having a large internal market, such as India, South Korea, and China, have taken steps in this direction. In India alone, there are now at least four hatchery organizations seriously involved in pureline breeding (Gaffar, 1986). The pureline hatcheries have imported pure or elite lines from overseas with the firm belief that sometime in the near future the government of India will stop the importation of breeding stocks.
7. Development of a breed by crossbreeding and backcrossing methods.

Development of native stock through purebreeding. Ahmed and Hashnath (1983) described the usefulness of native Deshi chickens in Bangladesh, which are usually scavengers, but which possess tremendous ability at surviving under stress conditions, resisting local diseases and retaining their scavenging habits. They produce 45 eggs in a laying cycle with an average egg weight of 33.5 g. The government of Bangladesh started a program for development of these chickens through supply of genetically improved Deshi cockerels obtained from the government breeding farm and hatchery at Dhaka. Continuous upgrading of the native females with the genetically improved males is expected to raise egg production and growth performance. The success of this scheme depends on the early slaughtering of unwanted males. A similar program has also been advocated by Leong and Jalaludin (1984) for development of village poultry in Malaysia, because the introduction of exotic breeds or strains into small farming systems demands a high level of management and expensive feed.

In the most poorly developed nations, the above breeding strategy would be quite appropriate, which means efforts need to be directed to the introduction and development of selection within local breeds. However, selection within breeds under extensive conditions will be extremely difficult due to lack of any genetic estimates of growth and reproduction parameters. Identification of birds in village conditions is also not easy. Hetzel (1983a, 1983b) argued that improvement of various local breeds of ducks in Indonesia could be beneficial, but evaluation of breeds and their crosses in specific environments is necessary before undertaking a breeding strategy.

Gradual replacement of local males. Genetically improved males of locally adapted exotic stock could be released by breeding farms to small-holder systems for village poultry development. This had been the basis of initial development in many countries. Research findings from Nigeria indicate that less productive local breeds can be of commercial and breeding utility if crossed with improved breeds or strains (Omeje and Nwosu, 1986). Their results showed that the body size and efficiency of F_1 crosses (local x Gold-link) could be as high as the imported Gold-link chickens, and the backcross progeny (F_1 x Gold-link) showed even higher growth performance and feed conversion efficiency without significant reduction in production traits. They suggested using continuous selection of the backcross progeny to develop a synthetic breed well adapted to Nigerian conditions. Similar suggestions were made by Prawirokusumo (1988) in Indonesia, and Coligado et al. (1986) in the Philippines. Theoretically, by a continuous upgrading scheme either through backcrossing or line breeding it is possible to obtain a synthetic breed genetically close to the imported breed, but research is needed to evaluate the comparative efficacy of the two methods.

Use of local genomes and major genes. According to Mérat (1979), about 12 loci carry genes influencing biological efficiency in chickens. Similar investigation has not been reported in other species of poultry. Horst (1982) tested these chicken loci for their productive adaptability in a warm environment, and suggested the use of these major loci in developing a strategy for poultry breeding in the tropics. Examples of the use of local genomes and major genes in tropical synthetic line breeding (Table 42.2) show that other major genes can be successfully introduced into local lines for genetic improvement and development of a new breed. Likewise, the benefits of introducing frizzle (*F*) and naked neck (*Na*) genes into local lines in the tropics for higher productive adaptability has been advocated by Horst (1982, 1988). Major effects of these genes on economic traits under Malaysian conditions have been recently investigated (Khadijah, 1988; Panandam, 1985; Mathur and Horst, 1988). Once their phenotypic effects on physiological and anatomical traits are characterized, it would be quite easy to integrate these genes into crossbreeding programs by establishing paternal breeding lines with dominant and/or sex-linked major genes. Many international breeding farms have already introduced dwarf lines for this purpose, and are investigating naked neck and other major genes (Flock, 1982).

Two-way crosses of breeds or lines. Almost all middle-level producers and invariably all large commercial enterprises in developing countries use hybrid chicken breeding stock, either originating from their government hatcheries or from commercial breeding farms. In the development and widespread utilization of intercrossing for commercial poultry production, two factors are of primary importance. Firstly, there is the phenomenon of heterosis which is expressed in the performance of hybrids, and secondly,

Table 42.2. Examples of the use of local genomes and major genes in the breeding of tropical synthetic lines. (Adapted from Horst, 1988).

Sire line	Dam line	Name and traits of synthetic line
Fayoumi	White Leghorn	Fay Horn
Fayoumi	Barred Plymouth Rock	Dokki 4
Barred Plymouth Rock	Fayoumi	Dokki 4
Silky	Rhode Island Red	Taiwanese dark meat
Silky	White Cornish	Taiwanese dark meat
Kadakanath	White Leghorn	Yamuna - dark meat, fast feathering, white plumage
Kadakanath	New Hampshire	Yamuna - dark meat, fast feathering, colored plumage

the selection of lines showing considerable genetic differences but good specific combining ability.

There have been many investigations on estimation of heterosis and combining abilities for economic traits in chickens. Cheong and Chung (1985) found two-way White Leghorn crossbreds had considerably higher hen-day and hen-housed egg production than purebreds. Heterosis among various crosses was up to 3.78 percent for hen-day production and up to 6.16 percent for hen-housed production. A heterotic effect of similar magnitude has been noted for other traits such as survival rate, age at sexual maturity, and body weight. Working with Alabio and Tegal ducks in Indonesia, Hetzel (1983a, 1983b) obtained heterosis percentages from crosses of these two breeds as follows: egg production percentage 7.4; egg mass 10.4; feed conversion ratio -9.9. Similar heterotic effects have been obtained in chickens by Jain and Chaudhary (1984) in India, by Ooi et al. (1975) in Malaysia, and by Soares and Fonseca (1984) in Brazil.

The favorable effects of heterosis have been established in crossing experiments involving imported pure lines and commercial lines as well. In many of these experiments, involving diallel crosses to estimate heterosis in specific two-way crosses, the general and specific combining ability of lines, heterosis and reciprocal effects were significant for body weight (Singh et al., 1983; Mukherjee, 1983). General and specific combining ability effects were also found to be significant in these studies for body weights at certain ages and for eviscerated weight.

Similar diallel cross experiments have been performed in White Leghorn lines to estimate heterosis for production traits. Jain et al. (1981) showed the occurrence of heterosis for egg production and sexual maturity in most of the two-way crosses within their diallel crosses of four lines. There is an abundance of literature on this type of work from India and South Korea, most of it being published in the *Indian Journal of Poultry Science* and the *South Korean Journal of Animal Science.*

In most developing countries, the government and private hatcheries will continue to import parent lines from international breeders, and their strategy will be to produce a two-way cross for commercial production. Stock from these local breeding programs will not be as efficient as the parent stock from the international breeders (Mukherjee, 1987).

However, for small-holder production systems, a local breeding program of two-way crosses could be quite useful. This requires maintenance of two breeds or two lines within a breed and selection within lines based on cross performance.

Four-way crosses. Current indications are that there is sufficient interest among some Brazilian and southeast Asian commercial breeders to upgrade their status from parent breeders to grandparent breeders. This trend will probably be followed by breeders of the Middle East and some other parts of South America.

A few elite nucleus flocks in different countries may attempt to develop new lines from imported commercial lines. Abplanalp (1986) suggested the possibility of developing inbred lines from hybrid crosses. But for less developed countries, similar objectives would be highly unrealistic because of the expense, labor and time involved. However, experiments at the University of Malaya (Mukherjee, 1983) showed high general and specific combining ability of some commercial lines when four male and four female parent broiler lines of four commercial companies (two American, one European, and one Australian) were crossed with each other using a diallel mating pattern; their progeny were found to show heterotic effects for broiler traits. It is assumed that crosses of the two best progeny groups will yield a commercial cross of significant importance. In countries with a large marketing potential, similar experiments may have been performed by the commercial sector, but their results have not been published.

Pure line selection. In large countries such as India, both government and industry recognize the growing demand for day-old chicks. Hence there are continuous efforts to develop pure lines for meat production and egg production locally, which may equal or excel in the future the best currently available (Rao, 1983, as cited by Mukherjee, 1987). Selection of lines for local adaptation and for ability to utilize locally available diets, and minimizing genotype x environment interaction effects, are some of the processes which may encourage local commercial breeders or government breeding farms to develop pure lines from imported pure lines or grandparent lines, or even certain breeds with relevant major genes. However, this needs a policy on the part of the government to stop importation, which will be difficult to make and to implement.

Development of a breed by crossbreeding. Abdel-Gawad et al. (1980) developed the White Mamourah breed of Egypt by crossing Alexandria males and inbred Dokki-4 females, and then backcrossing the F_1 females to Alexandria males. The backcrossed White Mamourah showed phenotypic superiority for the economic traits studied (body weight, breast width, and feed conversion ratio) over the F_1 and the parental breeds. Detailed description of morphology and color of this breed, which was genetically stabilized, was given.

Backcrossing procedures were also used by Arboleda et al. (1973) in crossing Vantress Dominant White (VDW) and Arbor Acres White Rock, then mating selected F_1 females to pure VDW males. The resulting backcross male and female progeny were selected for higher body weights at eight weeks of age and mated *inter se* for two generations. There was a ten percent increase in body weight of the selected group over the unselected backcrosses, and correspondingly an increase in percentage of first grade breast fleshing.

Similar linecrosses followed by backcrosses were made by Mukherjee (1983) using two commercial lines, but percentage increase of body weight among the backcrosses was not different than the F_1 performance.

Studies of this nature must have been made by many other commercial and government breeding farms with the objective of developing a new breed, but reports of these studies are not available, perhaps due to commercial secrecy.

SELECTION EXPERIMENTS IN DEVELOPING COUNTRIES

There is no dearth of selection experiments which have been conducted at government or institutional breeding farms in Asia. Published reports indicate research on several poultry species and a variety of topics.

Selection indexes and relative efficiency of selection methods. Selection on the basis of an index using data on body weight at eight weeks, egg production to 300 days, and percent hatchability, was found to be relatively more efficient than tandem selection or independent culling levels selection for all traits except hatchability. Index selection was 35 percent more efficient than selection on independent culling levels, which in turn was better than tandem selection (Sharma et al., 1983).

Comparisons have been made between index selection and mass selection for individual traits (body weights at eight and 20 weeks of age, 35-week egg weight, age at sexual maturity, and egg production to 260 days of age) by Verma et al. (1984) in White Leghorn strains. For aggregate genetic response, index selection was found to be 2.76, 3.33, 13.66, 1.32 and 1.53 times more effective than direct selection for egg production, egg weight at 35 weeks of age, initial egg weight, 20-week body weight and eight-week body weight, respectively.

Superiority of index selection compared to mass selection for broiler traits and for egg laying traits has been reported by other workers (Chung et al., 1983; Sang et al., 1983). Various selection indexes to improve egg production in White Leghorn flocks have been compared by Singh et al. (1984). When ten percent of the cockerels were selected in each population, selection of 40 percent of the females based on index selection was found to give better genetic response than when selection with different intensity was made for individual traits. Similar observations were noted by Ayyagari et al. (1985) in another White Leghorn population, and by Makarechian et al. (1983) while working with indigenous chickens of southern Iran.

There are various reports from India concerning the efficiency of selection based on part records of egg production (Ayyagari and Mohapatra, 1983; Ayyagari et al., 1980; Johri et al., 1985). In general, there is close agreement between observed and predicted genetic response to selection in all the experiments involving four generations of selection or more, which indicates that natural selection, genotype x environment interaction, and environmental fluctuations were unimportant during the course of selection. All these studies indicate that egg production records to 260-280 days of age would be sufficient for selection to increase total egg production in tropical locations.

Efficiency of one- and two-stage index selection for egg type chickens. Experiments conducted to investigate the comparative efficiency of one-stage selection of various traits with a two-stage selection index (where some traits were selected at an early age and some others at a later age) did show that single-stage indexes (where all the major traits are incorporated) had higher relative efficiencies compared to two-stage selection (Verma et al., 1984; Ayyagari et al., 1985). In the earlier study, the correlation between index and aggregate genotype ranged from 0.43-0.67 for various indexes, but it was always highest for the one-stage index using the major traits.

Restricted selection index in poultry breeding. Quite a few papers from India show the effect of a restricted selection index on maximizing genetic gain in egg production. One report (Narayanikutty and George, 1984) involving three restricted indexes, one on egg weight, another on 40-week body weight, and the third one with restriction on both these traits, showed that restriction of 40-week body weight would be more effective than the other restrictions.

Selection experiments in ducks. Brown Tsaiya and White Tsaiya were genetically improved by many generations of selection (Tai, 1986). A selection index to maximize genetic improvement of egg number to 40 weeks of age without reducing egg weight or increasing body weight was used within each of four strains of native Brown Tsaiya ducks. Their egg production performance to 35 weeks of age ranged from 105-111 eggs, with egg weight at 30 weeks ranging from 64.3-65.6 g. Selection in White Tsaiya started in 1966, mainly for completely white plumage and typical Tsaiya conformation. Four lines of White Tsaiya were developed: a control line without any major selection criteria except for a slight increase in egg numbers, a line selected for carcass appearance of mule ducks and to raise the value of the feathers, and two inbred lines with an inbreeding coefficient reaching not more than 50 percent and with a view to using these two inbred lines for future crossing to measure heterosis.

Selection for large body size and egg numbers in Pekin ducks, and selection in white muscovy ducks based on the white plumage of their mule progeny, has been also reported by Tai (1986). In Pekin ducks, increase in body weight due to selection resulted in lowered egg number and delayed sexual maturity. Muscovy sire lines were being selected to improve body weight and fertilizing capacity.

In India, Bulbule (1986) reported the construction of a selection index based on Osborne's index (1957), which is being used for the development of various strains of ducks kept at the Central Duck Breeding Farm, Hessarghata, India. Genetic progress in these strains as a result of selection has not yet been documented.

There is a wide variety of management systems for ducks in Asia: ducks herded on newly harvested rice, ducks on flooded fallow rice fields, ducks scavenging in dry fallow fields, ducks in ponds, canals and rivers, and ducks fully confined. Future selection objectives for these systems may have to be different (Mukherjee, 1986) compared to European selection objectives (Pingel and Heimpold, 1983; Powell, 1986). Powell (1986) presented evidence to suggest that commercial strains produced in Europe are gaining ground in the Asian markets, and eventually extensive systems of management will have to make way for modern enterprises. However, Mukherjee (1986) reported that for systems where egg production is more important than meat production, scavenging ducks will still play an important role, and selection for total number of eggs produced and persistency of production should be undertaken for future genetic gain. There have been many attempts to improve egg production by nongenetic means and crossbreeding (Hetzel, 1982, 1983a, 1983b), but faster genetic response could be obtained if crossbreeding is combined with selection of the native stock which is in use as one of the parental lines in the cross.

Selection experiments in other poultry species. There are some publications on selection for body weight and egg production in different lines of quail at the Central Avian Research Institute, Izatnagar, India (see Srivastava, 1987 for review). The relative efficiency of various selection methods for four-week body weight has been reported by Ahuja et al.

(1981), and for production efficiency by Sreenivasaiah et al. (1980). Literature on selection and breeding of turkeys, geese and guinea fowl in developing countries is not generally available, but it may exist in the regional literature, perhaps in native languages.

CONCLUDING COMMENTS

Chickens, ducks, quail, geese, turkeys, and guinea fowl are widely distributed in developing countries of the world. Their genetic improvement through breeding and selection within developing countries has just started. Present efforts are mainly restricted to chickens, and to a certain extent to ducks. However, the importation of strains of Western nations poses a threat to development of indigenous poultry breeds, since there is wide disparity between the performance of imported and native stocks, and this gap will increase.

Although the sales of commercial parent lines and perhaps later the grandparent lines produced in Western countries will increase, there should be greater effort on the part of planners in developing countries for making provision of proper identification, documentation, and evaluation of indigenous breeds of poultry species. Programs for conservation of genetic resources of domestic animals should therefore emphasize the conservation and development of native poultry.

Commercial breeding companies, mainly the international franchises, will probably design future breeding strategies that are appropriate for the dry and humid tropics. Greater emphasis has to be placed on disease resistance through immunogenetic methods, efficiency of feed conversion, heat tolerance, and desired plumage and shell color, in order to be competitive in many markets of developing countries.

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